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OPHTHALMOLOGICAL TRANSACTIONS

VOL. XXI

TRANSACTIONS

OF THE

OPHTHALMOLOGICAL SOCIETY

OF THE

UNITED KINGDOM

VOL. XXI

SESSION 1900-1901

WITH

LIST OF OFFICERS, MEMBERS, ETC.

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NOTICE.

The present volume comprises the proceedings of the Ophthalmological Society of the United Kingdom during its Twentieth Session, October, 1900, to July, 1901.

The Society does not hold itself responsible for the statements, reasonings, or opinions expressed in the communications which the Council has deemed suitable for publication.

11, Chandos Street.
Cavendish Square, W.
October. 1901.

TABLE OF CONTENTS

OF VOLUME XXI.

	PAGE
Officers and Council	IX
List of Presidents	X
List of Members	XI
Rules	XXX
List of Communications made during the Session 1900–1901	XLI
List of Plates	XLIX
List of Diagrams and Illustrations in the Text	L
Diseases of the Conjunctiva	1
Diseases of the Cornea	24
Diseases of the Iris and Ciliary Body	35
Sympathetic Disease	37
Diseases of the Crystalline Lens	43
Diseases of the Retina and Choroid	66
Parasites	89
Diseases of the Orbit and Sinuses	94
Injuries of the Eye and Orbit	98
Operations, Instruments, and Appliances	142
Congenital Conditions	165
Colour-Blindness	181
Histology	200
Pathological Anatomy	201
Miscellaneous	244
Appendix	312
Report of Council	315
Index	319

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O.M.—Original Member.
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V.-P.—Vice-President.
T.—Treasurer.
L.—Librarian.
S.—Secretary.
C.—Member of Council.
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†.—Denotes Non-Resident Life Members.

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1884 †Allison, H., M.D., care of Messrs. Binny and Co., Madras, India.

1899 Amenabar, Julio Daniel, Guayacan, Coquimbo, Chili.

1887 *Anderson, Tempest, M.D., 17, Stone-gate, York.

1883 Andrews, A. G., Carlton House, Moss Lane East, Manchester.

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1891 †Armstrong, Hugh, Hobart, Tasmania.

1895 Attlee, John, M.B., 58, Brook Street, W.

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1889 †Barrett, James W., M.D., 34, Collins Street East, Melbourne, Australia.

1883 Barton, J. Kingston, 2, Courtfield Road, Gloucester Road, S.W.

1892 Batten, Rayner D., M.D., Campden Lodge, Campden Hill Road, W.

1888 *Beaumont, W. M. (C.), 4, Gay Street, Bath.

O.M. *Beevor, C. E., M.D., 33, Harley Street, W. (S. 1891-4. C. 1894-7.)

1891 †Bennett, Alfred H., Kapunda, South Australia.

1897 Bennett, H. Percy, 12, Victoria Square, Newcastle-on-Tyne.

O.M. Benson, A. H., M.D. (V.-P.), 42, Fitzwilliam Square, Dublin. (C. 1888-91.)

O.M. Berry, G. A., M.D., 31, Drumsheugh Gardens, Edinburgh. (C. 1889-92. V.-P. 1895-8.)

1899 Bickerton, R. E., M.B., 137, Harley Street, W.

1881 *Bickerton, T. H., 88, Rodney Street, Liverpool. (C. 1895-8.)

1892 Black, John Wilson, M.D.Edin., 46, Academy Street, Inverness.

1891 Black, J. Urquhart, M.B., Taroom Hospital, Queensland, Australia.

1898 Blair, Charles Samuel, M.D., 117A, Harley Street, W.

1885 Blumer, W. P., 15, Esplanade West, Sunderland.

1890 Bokenham, T. J., 10, Devonshire Street, Portland Place, W.

ELECTED

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1897 Bower, John A., M.B., Portland House, Cheltenham.

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O.M. Brailey, W. A., M.D., 11, Old Burlington Street, W. (V.P. 1898-1901. S. 1883-6. C. 1880-3, 1886-9.)

1897 Breuer, August, M.D., 10, Finsbury Circus, E.C.

1899 Brewerton, Elmore, 19, Portland Terrace, Regent's Park, N.W.

1891 Bristowe, Hubert Carpenter, M.D., Wrington, Somerset.

1886 Bronner, Adolph, M.D., 33, Manor Row, Bradford. (C.)

1901 Brooks, R. Philip, 136, Tollington Park, N.

O.M. Browne, Edgar A., 39, Rodney Street, Liverpool. (C. 1887-90. V.-P. 1893-6.)

1898 Browne, James M., M.B., 27, Wellington Road, Cork.

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1901 Buchanan, Leslie, 17, Sandyford Place, Glasgow.

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1889 Bullar, John F., M.B., 7, Carlton Crescent, Southampton.

1883 †Buller, Frank, M.D., 838, Dorchester Street, Montreal, Canada.

1897 Bulteel, Marcus Henry, 84, Durnford Street, Stonehouse, Plymouth.

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O.M. Buzzard, Thomas, M.D., 74, Grosvenor Street, W. (V.-P. 1888-91. C. 1881-2.)

1897 Byers, W. Gordon M., M.D., Moorfields Eye Hospital, E.C.

1892 †Caiger, Herbert, M.B., Jacobsdal, Orange Free State.

1891 Campbell, E. Kenneth, M.B., 23, Wimpole Street, W.

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ELECTED

1882 *Cant, W. J., 3, Lindum Road, Lincoln. (C. 1898-1901.)

1891 Cargill, L. Vernon, 15, Stratford Place, W.

1888 Carter, Rowland W., M.D., 13, Gloucester Row, Weymouth, Dorset.

1895 Cartwright, E. H., M.D., 1, Bower Terrace, Maidstone, Kent.

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1886 Collins, W. J., M.S., M.D., B.Sc., 1, Albert Terrace, Gloucester Gate, Regent's Park, N.W.

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1898 Coulter, Robert J., M.B., Warwick House, Stow Hill, Newport, Mon.

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1898 Critchley, Harry G., M.D., St. Leonard's House, Banbury, Oxon.

1900 Cropper, John, Mount Ballan, Chepstow (Mon.).

1881 Cross, F. R., Worcester House, Clifton, Bristol. (V.-P. 1898-1901. C. 1891-4.)

1884 Davidson, James McKenzie, M.B., 76, Portland Place, W. (C. 1892-5.)

1889 †Davis, G. C., 3, Hyde Park Terrace, 173, Liverpool Street, Sydney, New South Wales.

1897 Dawnay, Arch. Hugh Payan, 48, St. Mary Abbot's Terrace, Kensington, W.

O.M. Dent, Clinton Thomas, 61, Brook Street, W.

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1890 Donaldson, Eben., 49, Great James Street, Londonderry.

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1890 Douty, Edward Henry, M.B., B.C., 69, Bridge Street, Cambridge.

1887 Doyne, Robert W., 64, St. Giles', Oxford. (C. 1892-5.)

1881 *Drake-Brockman, E. F., 14, Welbeck Street, Cavendish Square, W. (C. 1895-8.)

1889 †Drake-Brockman, Herbert E., I.M.S., care of Wm. Watson and Co., Bombay, India.

1895 Du Boulay, H. H., 2, Royal Terrace, Weymouth.

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1886 Dunn, Hugh Percy, 54, Wimpole Street, Cavendish Square, W.

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1900 Eason, Herbert L., M.B., Guy's Hospital, S.E.

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1900 Evans, Thomas, 211, Macquarie Street, Sydney, New South Wales.

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1896 Eyre, John W. H., M.D., Embankment Chambers, 19, Villiers Street, W.C.

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1890 Ferguson, R. Bruce, M.D., B.C., 26, Woodland Road, New Southgate, N.

1889 Ferrier, David, M.D., F.R.S., 34, Cavendish Square, W. (C. 1896-7.)

1894 Fischer, E. C., M.B., Royal London Ophthalmic Hospital, Moorfields, E.C.

1895 Fisher, J. Herbert, M.B., 83, Wimpole Street, Cavendish Square, W.

O.M. Fitzgerald, C. E., M.D., 27, Upper Merrion Street, Dublin. (V.P. 1882-5. C. 1880-1.)

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1886 Ford, A. Vernon, 14, High Street, Portsmouth.

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1887 †Gardner, John J., M.D., 12, Phillip's Place, Montreal, Canada.

1889 Gibbs, Alfred N. G., 52, Whiteladies Road, Clifton.

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1885 †Godfray, Alfred Charles, St. Heliers House, St. Heliers, Jersey.

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1897 Gordon, William, M.D., B.C., Barnfield Lodge, Exeter.

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1897 Granger, F. M., 18, Nicholas Street, Chester.

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1895 †Gray, James, M.D., 119, Robert Street, Toronto, Canada.

1887 Green, Edwin Collier, 27, Friar Gate, Derby.

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1895 Griffin, W. Watson, M.B., 68, Brunswick Place, Hove, Brighton.

1885 *Griffith, A. Hill, M.D., 17, St. John Street, Manchester. (C. 1893-6.)

1894 Grimsdale, Harold, M.B., 114, Harley Street, W.

O.M. Grossmann, K. A., 70, Rodney Street, Liverpool.

1899 Gruber, Rudolph, M.D., 67, Wimpole Street, W.

1889 Gunn, Donald S. (C.), 66, Wimpole Street, W.

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1887 Habershon, Samuel Herbert, M.D., 88, Harley Street, W. (S. 1894-7. C. 1897-1900.)

1889 Hailes, C. D. G., M.D., Hawkesdale, 27, Alma Road, Clifton.

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ELECTED

1900 Hallidee, Andrew, M.B., 8, Warrior Square Terrace, St. Leonards-on-Sea.

1900 Hamilton, John, 3 North Common Road, Ealing, W.

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1878 †Hamilton, T. K., M.D., Wakefield Street, Adelaide, South Australia.

1899 Hancock, W. Ilbert, Grove House, Belsize Grove, N.W.

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1886 Hartley, Robert N., M.B., 2, Clarendon Road, Leeds.

1882 *Hartridge, Gustavus, 12, Wimpole Street, W. (C. 1889-92. S. 1892-5. C. 1895-8.)

1899 Hawkes, C. S., c/o Dr. A. M. Hawkes, Glan-y-mor, Penally, Tenby, Wales.

1901 Hawthorne, C. O., 28, Weymouth Street, W.

1892 Haydon, Frank, Apothecaries' Hall, Water Lane, Blackfriars, E.C.

1898 Hayes, George C., 22, Park Place, Leeds.

1899 Henderson, Edward E., M.B., 12, Kensington Square, W.

1896 Henry, R. Wallace, M.D., 4, Market Street, Leicester.

1901 Henry, John P., M.D., 41, Welbeck Street, W.

1897 †Herbert, Major Herbert, I.M.S. (care of Messrs. H. S. King and Co., 45, Pall Mall, S.W.).

1887 Hern, John, M.D., Semmercote, Darlington.

1895 Hickman, H. R. Belcher, M.B., 5, Harley Street, W.

O.M. Higgens, Charles (V.-P.), 52, Brook Street, W. (C. 1880-3.)

1888 *Hinnell, J. S., M.B., 62, Garland Street, Bury St. Edmunds.

1899 Hobday, James, M.B., 12, Portland Gardens, Finsbury Park, N.

1886 †Hodge, Rev. Sydney Rupert, Wesleyan Mission, Hankow, China.

1897 Hogg, G. H., M.D., 95, George Street, Launceston, Tasmania.

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ELECTED

1898 Hopkinson, Emilius, 9, Holywell, Oxford.

1893 †Howe, Lucien, 183, Delaware Avenue, Buffalo.

1901 Hudson, Arthur A., 3, Ellerdale Road, Hampstead.

1884 †Hudson, Ernest, Central Gaol, Benares, N.W.P., India.

1889 †Hughes, Samuel H., Gingola, Mowbray Road, Willoughby, Sydney, New South Wales.

1893 †Hughes, Wilfrid Kent, M.B., 102, Collins Street, Melbourne.

O.M. Hutchinson, Jonathan, F.R.S., 15, Cavendish Square, W. (*Pres.* 1883-6. V.-P. 1880-1, 1886-9.)

1901 Inman, Wm., M.B., Royal Ophthalmic Hospital, City Road, E.C.

1883 †Jackson, James, M.D., Collins Street East, Melbourne, Australia.

O.M. Jackson, J. Hughlings, M.D., F.R.S., 3, Manchester Square, W. (*Pres.* 1889-90. V.-P. 1880-2, 1890-3.)

1898 James, George Brooksbank, 1, Carlisle Mansions, Victoria Street, S.W.

1888 James, J. T., M.D., 30, Harley Street, W.

1883 †Jenkins, E. J., M.D., Nepean Towers, Douglass Park, Sydney, N.S.W., Australia.

1883 Jessop, W. H. H., 73, Harley Street, W. (C. 1889-92.)

1882 Johnson, G. Lindsay, M.B., Cortina, 24, Netherhall Gardens, South Hampstead.

1888 †Johnston, Geo. D., Georgia Street, Vancouver, British Columbia.

O.M. Jones, Evan, Ty-mawr, Aberdare, Glamorganshire.

1898 *Jones, George, M.B., 8, Church Terrace, Lee, S.E.

O.M. Jones, H. Macnaughton, M.D., 141, Harley Street, W.

1897 Jones, Hugh E., 7, Rodney Street, Liverpool.

1894 †Jones, R. H., M.B., B.S., 209, Macquarie Street, Sydney, New South Wales.

O.M. Juler, H. E. (V.-P.), 23, Cavendish Square, W. (C. 1886-9.)

1899 Keeling, G. S., 23, Cavendish Square, W.

1900† Kelsall, H. T., M.D., 1, Devonshire Terrace, Perth, W. Australia.

ELECTED

1898 †Kendall, H. W. Martindale, Wellington, New Zealand.

1900 Kenna, Patrick James, M.B., Liverpool Street, Hyde Park, Sydney, N.S.W.

1888 †Kenny, Augustus Leo, M.B., 87, Collins Street, Melbourne, Victoria, Australia.

1890 Keogh, Alfred H., Surgeon-Major, M.D., M.Ch., Station Hospital, Barrackpore, Bengal, India.

1901 Kerr, James, M.D., 114, Manningham Lane, Bradford.

1891 Kingdon, E. C., M.B., C.M., 4, Upper College Street, Nottingham.

1900 Kirkland, Thomas Spier, M.B., College Street, Sydney, N.S.W.

1895 Knaggs, Robert Lawford, M.D., 27, Park Square, Leeds.

1881 †Knaggs, S. T., M.D., 16, College Street, Hyde Park, Sydney, New South Wales.

O.M. Lang, William (V.-P.), 22, Cavendish Square, W. (C. 1886-9.)

1881 Langdon, J. Winkley, 4, Winkley Square, Preston.

O.M. Lawford, J. B., 99, Harley Street, Cavendish Square, W. (C. 1886-9; 1898-1901. S. 1895-8.)

1889 *Laws, Wm. G., M.B., 3, East Circus Street, Nottingham.

1896 Lawson, Arnold, M.D., 12, Harley Street, W.

O.M. Lawson, George, 12, Harley Street, Cavendish Square, W. (C. 1882-4. V.P. 1892-5.)

1895 †Lea, J. Augustus, M.B., Royal Colonial Institute, Northumberland Avenue, W.C.

1885 †Le Cronier, Hardwick, St. Heliers, Jersey.

O.M. Lediard, H. A., M.D. (C.), 35, Lowther Street, Carlisle.

1885 Lee, Charles G., 11, Princes Avenue, Liverpool.

1896 Lister, W. T., M.B., Rose Hill, Totteridge, Herts.

O.M. Little, David, M.D. (*Pres.*), 21, St. John Street, Manchester. (V.-P. 1888-91. C. 1880-1.)

1892 Lodge, Samuel, jun., M.D., 13, Manningham Lane, Bradford.

ELECTED

1883 Lunn, J. R., Resident Medical Officer, Marylebone Infirmary, Notting Hill, W. (C. 1892-5.)

1899 Lyle, Herbert W., M.D.Lond., 178, Amhurst Road, Hackney Downs, N.E. (*Uncommunicated.*)

1892 Macarthur, Robert F., M.B.

1900 MacCallan, Arthur Ferguson, M.B., Royal London Ophthalmic Hospital, City Road, E.C.

1888* Mackay, George, M.D. (C.), 20, Drumsheugh Gardens, Edinburgh.

O.M. Mackenzie, F. M., 29, Hans Place, S.W.

1889 †Mackenzie, F. Wallace, M.B., 139, Upper Willis Street, Wellington, New Zealand.

O.M. Mackenzie, Stephen, M.D., 18, Cavendish Square, W. (S. 1880-2. C. 1882-5. V.-P. 1893-6.)

1889 MacLehose, Norman M., M.B., 13, Queen Anne Street, Cavendish Square, W.

1897 Maclennan, Duncan N., M.D., 596, Jarvis Street, Toronto, Canada.

1892 †MacLeod, Charles G., M.B., 26, College Street, Hyde Park, Sydney, N.S.W.

1881 †Maconachie, G. A., Brigade Surgeon Lieutenant-Colonel, M.D., 33, Queen's Road, Aberdeen.

1899 Maddox, Ernest E., M.D., Lansdowne Road, Bournemouth.

1883 †Maher, W. Odillo, M.D., 20, College Street, Hyde Park, Sydney, N.S.W.

1899 †Manché, Charles, B.A., M.D., 18, Sda. Moïni, Valletta, Malta.

1883 †Marlow, Frank William, M.D., 401, Montgomery Street, Syracuse, New York State, U.S.A.

1892 Marshall, Charles Devereux, 112, Harley Street, W.

1888 †Martin, Albert, M.D., Wellington, New Zealand.

1884 Maxwell, Patrick William, M.D. (C.), 19, Lower Baggot Street, Dublin.

1893 Maynard, Frederic Pinsent, Major, Indian Medical Service, care of Grindlay and Co. Calcutta.

ELECTED

1899 Maynard, George D., 7, Mount Park Crescent, Ealing, W.

1890 *McGillivray, Angus, M.D., 23, South Tay Street, Dundee, N.B.

O.M. McHardy, M. M., 5, Savile Row, W. (C. 1887-90.)

1895 †McIntosh, J. R., M.D., 44, King Square, St. John, New Brunswick, Canada.

1895 McKenzie, H. V., M.D., Elmbank, Abbey Road, Torquay.

1884 McKeown, David, M.D., 25, St. John Street, Manchester.

1884 McKeown, W. A., M.D., 20, College Square East, Belfast.

O.M. Meighan, T. S., M.D., 37, Elmbank Crescent, Glasgow.

1897 Miller, G. Victor, M.B., 2, Barrington Crescent, Stockton-on-Tees.

1899 Miller, Herbert Percy, M.D., 100, Stoke Newington Road, N.

1881 †Milles, W. Jennings, care of Drs. Henderson and Macleod, Shanghai, China.

1897 †Minnes, Robert Stanley, M.D., 127, Metcalfe Street, Ottawa, Ontario.

1896 Mooney, Herbert C., M.B., 22, Lower Baggot Street, Dublin.

O.M. Morton, A. Stanford, 133, Harley Street, W. (C. 1886-9.)

1898 Mott, F. W., M.D., F.R.S., 25, Nottingham Place, W.

1890 Mowat, Daniel, M.D., St. Ninian's, 93, Stamford Hill, N.

O.M. Mules, P. H., M.D. (V.-P.), Chester. (C. 1886-9.)

1899 Murphy, James Keogh, 35, Prince's Square, W.

1891 Myddelton-Gavey, Edward Herbert, 16, Broadwater Down, Tunbridge Wells.

1893 Napier, F. H., M.B., B.S., 18, Brandon Place, Glasgow.

O.M. *Nelson, Joseph, M.D., 29, Wellington Place, Belfast. (C. 1893-5.)

ELECTED

O.M. *Nettleship, Edward Nutcombe, Shottermill, near Haslemere. (*Pres.* 1895-7. S. 1880-3. C. 1883-6. V.-P. 1886-9.)

1881 Nicholson, A., 30, Brunswick Square, Brighton.

1895 *Ogilvie, F. Menteith, M.B., 53, Broad Street, Oxford.

1895 Ogilvy, Alexander, M.B., Montrose House, Clifton.

1889 †O'Kinealy, Frederick, Surg.-Capt. I.M.S., care of Messrs. King, Hamilton, and Co., 7, Hare Street, Calcutta.

1884 Oldham, Charles J., 38, Brunswick Square, Brighton. (C. 1897-1900.)

1899 Oldmeadow, Lloyd J. H., 15, Half Moon Street, W.

1881 Ormerod, J. A., M.D., 25, Upper Wimpole Street, W. (C. 1891-4.)

1899 Ormond, Arthur Wm., 22, St. Thomas Street, London Bridge, S.E.

1892 †Orr, Andrew William, M.D., A. M. P. Buildings, Edward Street, Brisbane.

1890 †Osborne, A. B., M.D., 46, McNab Street South, Hamilton, Canada.

O.M. Owen, D. C. Lloyd, 41, Newhall Street, Birmingham. (V.-P. 1891-4.)

O.M. Page, Herbert W., 146, Harley Street, W. (C. 1890-3.)

1890 †Palmer, L. Loran, M.D., 42, St. George Street, Toronto, Ontario, Canada.

1894 *Parker, Herbert, 7, Bloomsbury Place, W.C

1899 Parker, Herbert George, 29, Chorley New Road Bolton.

1900 Parsons, John Herbert, 28, Great Ormond Street, W.C.

1893 Part, J. Shepley, 20, Ashchurch Park Villas, Goldhawk Road, Ravenscourt Park, W.

1887 †Patell, D. H., 12, Ragoonatti Dadagi Street, Fort Bombay.

1900 Patkar, Bhagrant Sakharam, Esplanade Cross Road, Bombay, India.

ELECTED

1897 Pechell, Horace James, M.B., Alton House, Alton, Hants.

1888 Percival, Archibald Stanley, M.B., B.Ch., 26, Ellison Place, Newcastle-on-Tyne.

1891 Perry, A., Surgeon-Major, Principal Civil Medical Officer, Ceylon.

1889 †Perry, Francis F., Lahore Medical College, Punjab, India.

1889 Phillips, T., 124, Harley Street, W.

1895 Pickard, Ransom, M.D., 31, East Southernhay, Exeter.

1900 Pockley, Francis Antill, M.B., 227, Macquarie Street, Sydney, N.S.W.

1896 Pooley, G. H., Royal Westminster Ophthalmic Hospital.

1894 †Pope, R. J., M.D., Box 497, G.P.O., Sydney, N.S.W.

1900 Pope, Thomas Henry, M.D., B.Sc., Marshall's Road, Egmore, Madras, India.

O.M. Power, Henry, 37a, Great Cumberland Place, W. (*Pres.* 1890-3. V.-P. 1882-5, 1893-6. C. 1880-2.)

1899 Price, Henry J., Maldon, Essex.

1888 Price, John A. P., M.D., 124, Castle Street, Reading.

1882 Prichard, Arthur William, 6, Rodney Place, Clifton.

1892 Pronger, Charles Ernest, East Parade, Harrogate.

O.M. Purves, W. Laidlaw, 20, Stratford Place, Oxford Street, W.

1888 Rainy, Adam R., M.B., 6, Upper Wimpole Street, W.

1889 Ramsay, A. Maitland, M.D., 15, Woodside Place, Glasgow.

1899 Read, E. I., Government Medical Officer, Trinidad, West Indies.

1881 †Reeve, R. A., M.D., 22, Shuter Street, Toronto, Canada.

O.M. Reid, Thomas, M.D., 11, Elmbank Street, Glasgow. (V.-P. 1884-7.)

ELECTED

1885 Renton, James Crawford, M.D., 1, Woodside Terrace, Glasgow.

1891 Reynolds, Austin Edward, Highcroft, Shepherd's Hill, Highgate, N.

1897 Richmond, R., M.D., Brookfield, Woodside, Wimbledon, S.W.

1892 Ridley, Nicholas C., M.B., 27, Horse Fair Street, Leicester.

1900 Riseley, Stanley, Glossop Road, Sheffield.

1897 Rivers, W. H. R, M.D., St. John's College, Cambridge.

1885 *Roberts, Edward, 12, St. John Street, Deansgate, Manchester.

1896 †Roberts, J. R., Major, M.B., care of Messrs. King, King and Co., Bombay.

O.M. *Robertson, D. Argyll, M.D., 18, Charlotte Square, Edinburgh. (*Pres.* 1893-95. V.-P. 1881-2, 1886-9, 1896-9. C. 1895.)

1891 Robertson-Fullarton, Archibald Louis, M.B., C.M. 201, Bath Street, Glasgow.

O.M. Rockliffe, W. C., M.D. (V.-P.), 17, Charlotte Street, Hull. (C. 1892-5.)

1898 Roe, Arthur Legge, 45, Pryme Street, Hull.

1898 Roll, G. W., M.B., B.C., 7, Upper Wimpole Street, W.

1890 Rolston, John R., 14, The Crescent, Plymouth.

1891 Roper, Arthur Charles, The Shrubbery, Exeter.

1882 †Roth, Reuter E., 42, College Street, Hyde Park, Sydney, New South Wales.

1893 Rowan, John, M.B., 9, Blythswood Square, Glasgow, N.B.

1899 *Roxburgh, A. B., M.B., 7, Henrietta Street, W.

1881 †Rudall, J. T., 61, Spring Street, Melbourne, Australia.

1895 Russell, J. S. Risien, M.D. (C.), 4, Queen Anne Street, Cavendish Square, W.

1888 †Sanders, Richard C., M.D., 36, Chowringhee, Calcutta.

1884 *Sandford, Arthur W., M.D., 13, St. Patrick's Place, Cork. (C. 1896-9.)

ELECTED

1881 *Sansom, A. E., M.D., 84, Harley Street, W. (C. 1893-5.)

O.M. Savage, G. H., M.D., 3, Henrietta Street, Cavendish Square, W. (C. 1892-4.)

1900 Sayer, D. S., M.D., Brantford, Ontario, Canada.

1891 Schorstein, Gustave, M.D., 11, Portland Place.

1888 Scott, Kenneth, 7, Manchester Square, W.

1900 Scottneyliger, E. D., 89, Preston New Road, Blackburn.

1892 Shannon, John Rowlands, M.D., 18, West 35th Street, New York.

O.M. Sharkey, S. J., M.D. (V.-P.), 22, Harley Street, W. (S. 1885-8. C. 1888-91.)

1894 Shaw, Cecil E., M.D., 14, College Square East, Belfast.

1883 Shears, Charles H. (C.), 19, Upper Duke Street, Rodney Street, Liverpool.

1892 Sheppard, Arthur Murray, M.B., 2, Temple Gardens, E.C.

1883 Silcock, A. Q., 52, Harley Street, W. (S. 1889-92. C. 1892-5.)

1893 Simpson, R. J. S., M.B., care of Messrs. Holt and Co., Whitehall Place, S.W.

1899 Sinclair, A. H. H., M.B., 13, Walker Street, Edinburgh.

1891 Sinclair, Walter William, 16, Museum Street, Ipswich.

1898 Smith, G. Bellingham, M.B., 24, St. Thomas's Street, S.E.

1889 Smith, John, M.D., Brycehall, Kirkcaldy, N.B.

O.M. Smith, Priestley 34, Paradise Street, Birmingham. (V.-P. 1887-90; 1898-1901. C. 1883-6.)

1881 Smith, T. Gilbart, M.D., 68, Harley Street, W.

1900 Smith, William E., M.B., 47, Castle Street, Dumfries, N.B.

O.M. Snell, Simeon, 70, Hanover Street, Sheffield. (C. 1884-7. V.-P. 1892-5.)

1901 Snowball, Thomas, M.B., Royal Ophthalmic Hospital, City Road, E.C.

ELECTED

1889 Spencer, Matthew H., M.B., B.Ch., 95, St. Mark's Road, North Kensington, W.

1889 Spicer, Wm. T. Holmes, M.B. (C.), 47, Welbeck Street, W.

1897 Square, James Elliot, 22, Portland Square, Plymouth.

1895 †Stamberg, A. C., M.B., 5, Windsor Terrace, St. Heliers, Jersey, Channel Islands.

1886 Stephenson, Sydney (S.), M.B., 33, Welbeck Street, W. (C. 1898-1901).

1896 Stevenson, Edgar, M.D., 67, Rodney Street, Liverpool.

1893 Stirling, Alexander Williamson, M.D., Atlanta, Georgia.

1887 †Stirling, J. W., M.B., 873, Dorchester Street, Montreal, Canada.

O.M. Story, J. B., 6, Merrion Square North, Dublin. (C. 1885-8. V.-P. 1894-7.)

O.M. †Sturge, W. A., M.D., 15, Rue Longchamp, Nice, Les Alpes Maritimes.

O.M. Swanzy, H. R. (V.-P.), 23, Merrion Square North, Dublin. (V.-P. 1880-1, 1889-92. *Pres.* 1897-9.)

1888 *Sym, William George, M.D., 20, Alva Street, Edinburgh.

1883 †Symons, Mark Johnston, M.D., North Terrace, Adelaide, South Australia.

1886 Sympson, E. Mansel, M.D., Deloraine Court, Lincoln.

1898 Tait, Edward Sabine, M.D., 48, Highbury Park, N.

O.M. Tay, Waren, 4, Finsbury Square, E.C. (C. 1880-2.)

1882 Taylor, C. Bell, M.D., Beechwood Hall, Mapperley Park, Notts.

1899 Taylor, Inglis, M.B., 24, Wimpole Street, W.

1891 Taylor, James, M.D. (C.), 49, Welbeck Street, W. (C. 1894-7. S. 1898-1900.)

1889 Taylor, S. J., M.B., 34, Prince of Wales' Road, Norwich.

1900 Teale, Michael A., 38, Cookridge Street, Leeds.

O.M. Teale, T. Pridgin, F.R.S., 38, Cookridge Street, Leeds. (V.-P. 1880-1.)

ELECTED

1909 Thomas, Frank G., M.B., 2, Brunswick Place, Swansea.

O.M. Thomas, Jabez, Ty-Cerrig, Swansea.

1895 Thompson, A. Hugh, M.D., New Cavendish Street, W.

1885 Thompson, C. Sinclair, The Quay, Bideford, Devon.

1895 Thompson, George W., M.B., 27, Queen Anne Street, W.

1888 Thompson, John Tatham, M.B., (C.), 24, Windsor Place, Cardiff.

1895 Thompson, Robert, M.D., B.S., Dalkeith House, Wharf Street, Brisbane.

1898 Thomson, W. Ernest, M.D., 17, Berkeley Terrace, Glasgow.

1883 †Tobin, William, 31, Hollis Street, Halifax, Nova Scotia, Canada.

1900 Tomlinson, John H. Belmont, Vicarage Road, Egham.

1895 Toombs, Herbert George. 18, Faraday Mansions, Queen's Club Gardens, West Kensington.

1883 Tooth, Howard H., M.D., 34, Harley Street, W. (C. 1897-1900.)

O.M. Tosswill, L. H., 28, West Southernhay, Exeter. (C. 1896-9.)

1890 Turner, William Aldren, M.D.(S.), 13, Queen Anne Street, W. (C. 1897-1900.)

O.M. Tweedy, John, 100, Harley Street, W. (C. 1884-7. V.-P. 1891-4.)

1898 Tyrrell, F. Astley Cooper, M.B., Royal London Ophthalmic Hospital, City Road, E.C.

1883 Uhthoff, J. C., M.D., Wavertree House, Brunswick Place, Brighton.

1894 Usher, C. H., M.B., 3, Bon Accord Square, Aberdeen.

1888 Walker, Cyril H., M.B., 16, Pembroke Road, Clifton, Bristol.

O.M. Walker, G. E., 45, Rodney Street, Liverpool.

1892 Walker, H. Secker, 44a, Park Square, Leeds.

1901 Walmisley, Nicolas, 12, Station Road, Manor Park, Essex.

ELECTED

1900 Wardale, John D., M.B., 8, Eldon Square, Newcastle-on-Tyne.

1893 Warner, F. Ashton, 10, Brechin Place, S.W.

1893 Warren, H. Guy S., 205, Macquarie Street, Sydney.

1893 †Weekes, Charles Jones, 36, College Street, Hyde Park, Sydney.

1887 Wells, Arthur P. L., M.B., 83, Harley Street, W. (C. 1896-9.)

1885 Werner, Louis, M.B., 31, Merrion Square North, Dublin.

O.M. West, Samuel, M.D., 15, Wimpole Street, W. (C. 1888-91.)

O.M. *Wherry, G. E., M.B. (C.), Corpus Buildings, Cambridge.

1895 Whitehead, Arthur Longley, M.B., 4, Park Square, Leeds.

O.M. Williams, R., 82, Rodney Street, Liverpool. (V.-P. 1896-9.)

1894 Williams, W. E., M.B., Portmadoc, Carnarvonshire.

1888 †Willis, C. Fancourt, M.D., Mahableshwar, India.

1900 Wood, C. G. Russ, Hardwick House, Shrewsbury.

1889 Wood, David J., M.B., 41, Grave Street, Cape Town, South Africa.

1889 Woodburn, William Harrison, Kent Place, Ulverston.

O.M. Woodhead, G. Sims, M.D., 6, Scrope Terrace, Cambridge. (C. 1894-5.)

1899 Worth, Claud, 138, Harley Street, W.

1890 *Wray, Chas., Bank Chambers, North End, Croydon.

1898 †Wright, Edward Wm., M.D., 115, Montague Street, Brooklyn, New York.

1896 Yarr, M. T., Major, R.A.M.C., c/o the Siamese Legation, 23, Ashburn Place, S.W.

RULES.

1. Object of the Society.—The object of the Society is the cultivation and promotion of Ophthalmology in the United Kingdom, India, and the Colonies.

2. Constitution.—The Society shall consist of Ordinary and Honorary members. All registered medical practitioners of the United Kingdom, and all legally qualified medical practitioners in India and the colonies, whose qualifications are satisfactory to the Council of the Society, shall be eligible as ordinary members.

3. The officers of this Society shall consist of a President, four or more Vice-Presidents, a Treasurer, two Secretaries, a Librarian, and twelve other members, who together shall form the Council and manage the Society's affairs.

4. Ordinary Members.—Candidates shall be proposed on a form provided for the purpose and signed by three members from personal knowledge. The proposal paper shall be read at one Ordinary Meeting, and the Ballot shall be taken at the following meeting. No election shall take place unless ten members vote, and no person shall be elected who does not obtain four fifths of the votes given.

5. Form of Admission by the Chairman.—Members shall be admitted personally by the following form, after signing their names in the Admission Book, and paying their first annual Subscription. *Form of admission.*—"By the authority and in the name of the Ophthalmological Society of the United Kingdom I admit you a member thereof."

6. Honorary Members.—The Council shall have the power of proposing for election as Honorary members men of dis-

tinguished eminence in Ophthalmology, or in the sciences bearing upon it. They shall be elected by ballot in the same manner as Ordinary members at the next meeting. The Honorary members shall not exceed ten in number.

7. Resignation of Members.—Any member may retire from the Society after giving notice in writing to the Secretaries and paying any contribution due.

8. Re-admission of Members.—Any member who has retired from the Society and wishes to rejoin it, must be proposed, balloted for, and admitted in accordance with Rules 4 and 5.

9. Expulsion of Members.—A member can be expelled only at a General Meeting especially called for that purpose, and of which a written notice shall have been sent to every member at least fourteen days previously. At least ten votes must be recorded, and four fifths shall carry the expulsion.

10. Subscriptions.—The Annual Subscription shall be One Guinea, payable in advance in the month of October. Each member on election shall pay an Entrance Fee of One Guinea in addition to the Subscription, but in the case of a member elected at a meeting of the Session subsequent to Easter such member shall not be required to pay a Subscription during the next Session.

11. Arrears.—Any member whose Subscription is six months in arrear shall be reminded of the same by one of the Secretaries, and shall cease to be a member if it be not paid within the current year.

12. Composition Fee for Resident Members. — Any member may on entrance pay a Composition Fee of Fifteen Guineas, and be thereby exempted from paying any further Subscriptions, such member enjoying all the rights and privileges of a Subscribing member. After entrance, the Composition Fee will be according to the following scale:— After less than five payments Fourteen Guineas, after five payments Thirteen Guineas, after ten payments Twelve Guineas, after fifteen payments Eleven Guineas, after twenty payments Ten Guineas, after twenty-five payments Eight and a Half Guineas, after thirty payments Seven Guineas, after thirty-five payments Five Guineas, after forty payments Two and a Half Guineas, and after forty-five payments *nil*.

13. Composition Fees for Non-Resident Members.—Any member resident out of the United Kingdom may pay a Composition Fee of Seven Guineas instead of the Annual Subscription, and will then be entitled to receive, post free, a copy of the Society's 'Transactions' each year; but if at any time such member subsequently become a Resident member of the Society, payment of the Annual Subscription shall be resumed at the end of six years from the date of election, or if more than six years have elapsed, from the date of return to residence. The question of further composition shall be decided by the Council. N.B.—The Composition Fee will be held to include the Entrance Fee if paid any time during the First Session.

14. Election of Officers.—The Officers of the Society shall be elected yearly by Ballot at the Annual Meeting. A Balloting list of the names recommended by the Council for election shall be sent to each Resident member, together with the notice of the Annual Meeting. In the event of any name being substituted for any recommendation of the Council, the election shall not be valid unless effected by a majority of those present at the meeting. The President shall not hold office for a period of more than two consecutive years. No other member of the Council, except the Treasurer and the Librarian for the time being, shall hold the same office for more than three consecutive years. Any ordinary member of Council who has been absent from all the meetings of the Council for one session shall not be eligible for election to the Council for the ensuing session.

15. Scrutineers.—Two Scrutineers appointed by the President at the commencement of the Annual Meeting shall receive the lists during the first hour, and report the result to the President, who shall have a casting vote.

16. President and Vice-Presidents.—The President shall regulate all the proceedings of the Society and Council, state and put questions, interpret the application of the laws, and decide any doubtful points. He shall check irregularities and enforce the observance of the laws. He shall sign the minutes of General and Council Meetings. In the absence of the President one of the Vice-Presidents, the Treasurer, or some other member chosen by the meeting, shall perform his duties.

17. Secretaries.—The Secretaries shall manage all correspondence, shall attend every meeting of the Society and Council, and take minutes, which shall be read at the following meeting. They shall notify to new members their election. They shall arrange with the President the order of proceedings at all the meetings. They shall have charge of and keep a register of all papers communicated, and shall be the Editors of the 'Transactions.'

18. Treasurer.—The Treasurer shall receive all moneys due to the Society, and make all payments ordered by the Council, keeping an account of all such receipts and payments. He shall keep a printed receipt book for the subscriptions, and every receipt shall be signed by himself and countersigned by one of the Secretaries. He shall present to the Annual Meeting a written Report of the financial state of the Society, signed by himself and by two members of the Audit Committee.

19. Librarian.—The Librarian shall have entire charge and control of the Library. He shall purchase books for the Library as opportunities arise at his discretion out of the grant previously voted for this purpose by the Council. He shall see that all books belonging to the Society are duly entered in the Catalogue, and that the periodicals and pamphlets are from time to time, as occasion may require, suitably bound. It will be his duty to see that the Library Rules are not infringed.

20. Audit Committee.—The President, one of the Secretaries and two members of the Society nominated by the President at some meeting of the Society previous to the Annual Meeting, shall form a Committee to audit the Treasurer's accounts.

21. Meetings of Council.—The Council shall meet immediately after the meetings in October, January, May, and June, and at such other times as they may be specially convened. Three shall form a quorum. The Council shall determine questions by show of hands (or by Ballot if demanded), the President having in both cases a casting vote in addition to his ordinary vote. The Council shall decide upon all questions relating to the reception of communications, and to their publication in the Society's 'Transactions.'

22. Vacancies of Officers.—The Council shall have the power of filling up any vacancies which may occur in any of

the offices of the Society between one Annual Meeting and another.

23. 'Transactions.'—A copy of the 'Transactions' shall be sent to each Ordinary member of the Society whose subscription is not in arrear, and a copy of the 'Transactions' shall be sent to each Honorary member of the Society.

24. Meetings.—These shall consist of Ordinary, Clinical, Special, and General Meetings.

Ordinary Meetings.—The business during the first half-hour shall consist of the discussion of cases and card specimens, after which papers shall be read and discussed. Nothing relating to the laws or management of the Society shall be considered.

Clinical Meetings.—One or more of the meetings in each session may be devoted exclusively to the exhibition and discussion of cases and specimens. The number and dates of such meetings shall be arranged by the President and Secretaries.

The bye-laws and regulations relating to Ordinary and Clinical Meetings will be found on page xxxviii.

Special Meetings.—At the discretion of the Council an extra meeting may be arranged in order to hold a discussion upon some subject, of which due notice shall be given, or one of the Ordinary Meetings of the Society may be devoted to this purpose.

General Meetings.—The Annual General Meeting, of which every resident member shall receive one week's notice, shall be convened by special summons; ten shall form a quorum. It shall be held immediately after the Ordinary Meeting in July. The business shall consist in the election of Officers, the presentation and adoption of the Annual Report, and the discussion of any proposed alteration of the Rules, notice of which shall be given in the summons convening the meeting. No alteration in the Rules shall be adopted unless four fifths of the votes are given in its favour.

A Special General Meeting may be called at any time, on one week's notice, by the President or any three members of the Council, or on the requisition of fifteen members of the Society. The nature of the business to be transacted at such meetings

shall be specified in the summons sent to each member of the Society, and no other business shall be considered.

Dates and Hours of Meetings.—The dates of meetings shall be, unless otherwise determined by the Council, the third Thursday in October, the second Friday in November and second Thursday in December, the last Thursday in January, the second Friday in March, the first Thursday in May, the second Thursday in June, and the Friday after the first Thursday in July. All but the General, Clinical, and Special Meetings shall commence at 8.30, and shall not be prolonged after 10 p.m., except on the proposal of a member, duly seconded, and carried by a show of hands. The hour of all other meetings shall be determined by the President and Secretaries.

25. Visitors.—Each member may introduce as visitors two members of the medical profession to all but the General Meetings. They shall sign their names in the attendance book, opposite to the name of the member introducing them.

26. Order of Communications.—Communications shall be taken in the order in which they have been sent in to the Secretaries, subject to the discretion of the President. If an author be not present when the time arrives for his communication to be read, it shall be dealt with as the President may direct.

27. Publication of Papers and Discussion.—All papers must be sent to the Secretaries at least one week before the meeting, together with an abstract suitable for immediate publication in the journals. When possible, notice should be given relating to card cases, etc. All papers read before the Society shall be deemed the exclusive property of the Society, and if published elsewhere by the author without the express permission of the Council shall thereby be disqualified for admission into the Society's 'Transactions.' No report of the meetings of the Society may be published by members or others without the sanction of the Council.

28. Committee of Reference.—Six members of the Society shall be chosen annually by the Council to act with the Secretaries as a Committee of Reference, and any communication to the Society may be referred by the Secretaries to two or more members of such Committee of Reference for the

purpose of considering the fitness of the communication in question to be read before the Society, or to be published in the Society's 'Transactions.'

LIBRARY RULES.

1. The Library shall be open at the same hours as that of the Medical Society, viz. from 1 p.m. to 6 p.m. daily, except on Saturdays, when it will be closed at 3 p.m.

2. Members will be entitled to read the books belonging to the Society at 11, Chandos Street, between those hours, or to take them out on signing a book provided for that purpose. But any books of extraordinary value may be placed by the Council on a separate list, such books not being allowed to be removed from the Library.

3. A large number of the current periodicals will be accessible to members in the Library. These will not be allowed to be taken out of the Library.

4. A book must be returned at the expiration of a fortnight if wanted by any other member. The Librarian will in such a case write to the member in whose name the book was taken out.

5. If the book be not returned within four days of such notice a fine of 6d. will be charged for each day that the book is retained beyond such days of grace.

6. Instruments and drawings cannot be taken out of the Library except with the express permission of the Council.

7. A member taking out a book will be held responsible for its being returned in good condition.

THE BOWMAN LECTURE.

Resolution of Council, September 18th, 1883.

"That in recognition of Mr. Bowman's distinguished scientific position in ophthalmology and other branches of Medicine, and

in commemoration of his valuable services to the Ophthalmological Society, of which he was the first President, the Council shall each year, or periodically, nominate some person to deliver a lecture before the Society, to be called 'The Bowman Lecture,' which shall consist of a critical *résumé* of recent advances in ophthalmology or in such subject or subjects as the Council shall select, or of any original investigation, and shall be delivered at a special meeting of the Society held for the purpose, at which no other business shall be transacted."

BYE-LAWS CONCERNING COMMUNICATIONS.

1. The 'Transactions' shall consist of such communications made to the Society by or through members, as may be deemed by the Council suitable for publication. Also of discussions of importance or interest arising out of such communications.

2. No communication to the Society shall occupy more than twenty minutes, and in the subsequent discussion of it no member shall speak more than once, or for more than ten minutes, without the special permission of the Chairman.

3. All communications accepted by the Society become the property of the Society.

4. Communications are admissible which may have been read elsewhere, provided they have not been published, and are not intended to be published, in whole or in abstract, through another channel.

5. The cost of illustrations shall be borne by the Society so far as, in the opinion of the Council, is consistent with the state of its funds.

6. Reprints of papers may be obtained by authors at their own expense, by arrangement with the printer.

REGULATIONS CONCERNING THE EXHIBITION OF PATIENTS AND OF PATHOLOGICAL SPECIMENS BY CARD.

A. Patients must attend not later than 8 p.m., and will be allowed to leave at 9.30. A card, provided by the Society, must be placed conveniently near the patient (unless it is undesirable that it should be read by the patient or friends), and on it must be clearly written an account of the case, *comprising all the particulars intended for publication.* The title only of the case will be announced by the President to the meeting, but the Exhibitor (or his representative) must be present at the meeting, and be willing to read the case and furnish additional details if called upon to do so; the length of such oral communications not to exceed five minutes.

The narration and discussion of Card Specimens shall not occupy more than the first half-hour of the meeting.

B. Pathological Specimens may, at the discretion of the Exhibitor, be shown by card, and will then be subject to the above regulations. It is particularly to be noted that the description on the card must *comprise all the particulars intended for publication.*

REGULATIONS FOR CLINICAL EVENINGS.

1. Notice of cases or specimens should be sent in to the Surgical Secretary as early as possible, but not later than the Thursday preceding the meeting.

2. Cases and specimens may be shown without previous notice if there is time, but not until all on the printed list have been disposed of.

3. The number of cases to be shown by any member, and the total number on one evening to be left to the discretion of the President and Secretaries.

4. A list of the cases and specimens to be sent before the meeting to every member in the kingdom.

5. Particulars of each case to be fully written out before the meeting, and given to the Secretaries at the close of the meeting.

6. A short abstract of the case to be written on the card provided for the purpose, and placed by the patient.

7. The narration of a case shall not occupy more than five minutes, and in the subsequent discussion no member shall speak more than once or for more than five minutes.

8. Patients and specimens to be in place and ready by 8 p.m., one of the Secretaries being in attendance.

9. That the meeting for the narration and discussion of cases shall commence at 9 p.m.

10. If the exhibitor or a representative be not present the case cannot be taken.

11. A case which has been shown at one of the Ordinary Meetings may be subsequently brought forward at a Clinical Evening.

LIST OF COMMUNICATIONS

BROUGHT BEFORE THE SOCIETY DURING THE SESSION 1900-1901.

I. DISEASES OF THE CONJUNCTIVA.

		PAGE
1.	Tuberculosis of conjunctiva. By J. Herbert Fisher	1
2.	Primary chancre of retro-tarsal fold of upper lid. By Arthur W. Ormond	3
3.	Conjunctival growth (? pinguecula). By W. C. Rockliffe, M.D.	5
4.	Sequel to case of tuberculosis of conjunctiva, shown July 6th, 1900 ('Transactions,' vol. xx, p. 51). By W. H. Jessop	7
5.	New growth on conjunctiva and cornea. By W. Lang	8
6.	Notes of a case of membranous conjunctivitis. By J. F. Bullar	9
7.	Spring catarrh in the quiescent stage. By H. T. Holmes Spicer	12
8.	Further notes of a case of melanotic sarcoma of the conjunctiva. By J. B. Lawford	13
9.	Conjunctival bridges and pouches; union of retro-tarsal folds. By Herbert Herbert, I.M.S., Bombay	16

II. DISEASES OF THE CORNEA.

PAGE

1. Epithelioma of cornea. By Simeon Snell, with pathological report by E. Treacher Collins 24
2. Growth on cornea. By A. Stanford Morton 27
3. Epibulbar sarcoma. By J. Herbert Fisher 29
4. Spreading opacity of the cornea following herpes frontalis. By W. T. Lister 31
5. Double symmetrical opacities of cornea (rare form) removed by operation. By P. H. Mules, M.D. 32

III. DISEASES OF THE IRIS AND CILIARY BODY.

1. Serous iritis with nodular formations in the anterior chamber. By Rayner D. Batten 35
2. Serous iritis with persistent tachycardia. By Rayner D. Batten 36

IV. SYMPATHETIC DISEASE.

Wound of eyeball with prolapse of iris; enucleation of eyeball twenty days after the accident; followed fifty-four days later by sympathetic disease of the other eye; recovery. By Sydney Stephenson, M.B. 37

V. DISEASES OF THE CRYSTALLINE LENS.

1. Ætiology of lamellar cataract. By Norman G. Bennett (introduced) 43

VI. DISEASES OF THE RETINA AND CHOROID.

PAGE

1. Superficial choroidal atrophy, without subjective symptoms, in a member of a family subject to night-blindness. By A. Hugh Thompson, M.D. 66
2. An unusual form of choroiditis. By Charles Blair 68
3. Injury to an eye by direct sunlight, "eclipse blinding," with obstruction of a retinal artery and hæmorrhage into the vitreous. By Rayner D. Batten 70
4. A case of central choroiditis, simulating a neoplasm, watched for fourteen years. By Gustavus Hartridge 80
5. A case of choroiditis shown in an early stage in December, 1898. By Gustavus Hartridge 81
6. Nævoid condition of retina. By H. Work Dodd 82
7. Nævoid condition of the vessels of the disc, with nævus of the eyelid and face. By Gustavus Hartridge 83
8. Subretinal cystic swelling at centre of fundus. By J. Herbert Fisher 83
9. Secondary carcinoma of both eyes following scirrhus of breast. By W. C. Rockliffe 84
10. Recent central choroidal disease of unusual appearance (? tuberculous). By R. Marcus Gunn 87
11. "Hole" at the macula. By N. B. Harman, M.B. 88

VII. PARASITES.

A case of intra-ocular hydatid. By David J. Wood, M.D. (Capetown), with a postscript by E. Nettleship 89

VIII. DISEASES OF THE ORBIT AND SINUSES.

PAGE

1. Double frontal sinus mischief simulating symmetrical exostoses in the neighbourhood of the lacrymal sac.
By John Griffith 94
2. Case of probable frontal mucocele.
By Walter H. Jessop 95

IX. INJURIES OF THE EYE AND ORBIT.

1. Rupture of the lamina cribrosa and optic nerve-fibres at the papilla. By William Lang. 98
2. Diminished tension and the appearances of optic neuritis persisting for nine months after a wound of the orbit by a shot from an air-gun.
By E. Treacher Collins and J. S. Hinnell, M.D. 100
3. Cases of blindness from gunshot injury of the orbit.
By E. Nettleship 102
4. Case of bullet wound of orbit followed by blindness of both eyes.
By E. Treacher Collins and A. Vernon Ford. 121
5. Concussion injuries of the left eyeball.
By J. B. Lawford 122
6. A case of bullet injury of the occipital lobes, with loss of the lower half of each visual field.
By Anderson Critchett 123
7. Bullet wound of occipital region.
By J. Herbert Fisher 127
8. Case of pulsating exophthalmos, following injury to the head.
By D. J. Wood (communicated by E. Nettleship) 135
9. Two cases of eye injury due to lightning.
By M. T. Yarr, R.A.M.C. (South Africa) 139

X. OPERATIONS, INSTRUMENTS, AND APPLIANCES.

PAGE

1. A diagrammatic model intended to assist in the teaching of ocular refraction. By H. Secker Walker 142
2. A capsulotomy knife. By Gustavus Hartridge 143
3. Recording colour perimeter. By J. H. Tomlinson 144
4. On peritomy in episcleritis, iritis, herpes ophthalmicus, and purulent ophthalmia. By T. Pridgin Teale 146
5. A portable Refractometer. By C. Blair 156
6. Thiersch grafting for xerophthalmia. By Arnold Lawson 157
7. An apparatus for the performance of vibration massage, as adopted for the treatment of eye diseases. By Ainslie Hudson 158
8. New pattern of eye speculum. By H. E. Drake-Brockman 162

XI. CONGENITAL CONDITIONS.

1. Congenital malformation of eyelids with symmetrical lacrymal fistulæ. By Harold B. Grimsdale 165
2. A case of congenitally imperfect separation of iris from back of cornea; rise of tension induced by use of a mydriatic. By J. Herbert Fisher 166
3. A rare example of persistent capsulo-pupillary membrane. By G. Brooksbank James 168
4. A large area of retinal pigmentation below the disc. By G. Brooksbank James 169
5. Coloboma of optic disc (left). By J. Herbert Parsons 171
6. A case of high hypermetropia. By H. Work Dodd 171
7. Dermoid of conjunctiva with fissure of lid. By William Lang 172

PAGE

8. Coloboma of eyelid with deformity of auricle and dermoid growth. By W. Adams Frost 173
9. Two cases of congenital ptosis upon which a modified Hess's operation had been performed. By G. W. Roll 174
10. Congenital fibro-vascular tumour of eyeball. By F. R. Cross 178
11. Double coloboma of right lens. By A. Stanford Morton 179

XII. COLOUR-BLINDNESS.

1. A trichromic or three-unit case of colour-blindness. By F. W. Edridge-Green, M.D. 181
2. The evolution of the colour-sense. By F. W. Edridge-Green, M.D. 182

XIII. HISTOLOGY.

Sections showing opaque nerve-fibres in the retina. By Percy Flemming 200

XIV. PATHOLOGICAL ANATOMY.

1. Microscopical specimen of an osteosarcoma of the orbit. By John Griffith 201
2. Hypertrophy and ossification of tarsus. By Herbert Herbert 202
3. An encapsulated sarcoma of the right orbit. By Simeon Snell 204
4. Case of carcinoma of the corneo-scleral margin. By Leslie Buchanan, M.D. 205

PAGE

5. Note on a streptothrix mass (? actinomyces) removed from the right inferior canaliculus.
By Geo. Mackay, M.D. 207
6. Cyclitis: a study of inflammatory exudations into the vitreous body in cases of cyclitis.
By Leslie Buchanan, M.D. 208

XV. MISCELLANEOUS.

1. Voluntary bilateral nystagmus. By H. Work Dodd 244
2. The orthoptic treatment of convergent squint in young children. By C. Worth 245
3. The determination of the influence of the eyeball on the growth of the orbit, by experimental enucleation of one eye in young animals.
By W. Ernest Thomson, M.D. 258
4. On so-called paresis of divergence.
By G. A. Berry, M.B. 268
5. Note on two rare forms of non-paretic, non-concomitant squint. By G. A. Berry, M.B. 284
6. Double temporal hemianopia with slight optic neuritis and hemianopic pupil reaction; ? hæmorrhage into optic nerve sheath at chiasma; ? gumma.
By A. Hugh Thompson, M.D. 286
7. Paralysis of third nerve with unusual complications.
By P. H. Mules, M.D. 292
8. On Erythropsia. By W. H. R. Rivers, M.D. 296
9. A case of blindness resulting from the administration of salicylate of sodium. By Simeon Snell 306
10. A note with regard to the surface of the normal cornea.
By H. Herbert (Bombay) 308

XVI. APPENDIX . . 312

Report of Council 315
Index 319

LIST OF PLATES.

PLATE		FACING PAGE
I.	Conjunctival bridges and pouches. (Major H. HERBERT.) (Page 16) .	16
II.	Fig. 1. An unusual form of choroiditis. (CHARLES BLAIR.) (Page 68) .	70
	Fig. 2. Injury to an eye by direct sunlight. (RAYNER D. BATTEN.) (Page 70)	
III.	Fig. 1. A case of choroiditis. (GUSTAVUS HARTRIDGE.) (Page 81) . .	82
	Fig. 2. Nævoid condition of retina. (H. WORK-DODD.) (Page 82) . .	
IV.	Fig. 1. Recent central choroidal disease of unusual appearance. (R. MARCUS GUNN.) (Page 87) . .	87
	Fig. 2. A large area of retinal pigmentation below the disc. (G. BROOKSBANK JAMES.) (Page 169) . .	
V.	Figs. 1 and 2. Rupture of the lamina cribrosa and optic nerve-fibres at the papilla. (WILLIAM LANG.) (Page 98)	98
VI.	Figs. 1 and 2. A rare example of persistent capsulo-pupillary membrane. (G. BROOKSBANK JAMES.) (Page 168)	168
	Figs. 3, 4, and 5. Congenital fibro-vascular tumour of eyeball. (F. R. CROSS.) (Page 178) . .	
VII.	Figs. 1 and 2. Double coloboma of right crystalline lens. (A. STANFORD MORTON.) (Page 179) . .	180
VIII—XVI.	Cyclitis: a study of inflammatory exudations into the vitreous body in cases of cyclitis. (LESLIE BUCHANAN.) (Page 208) . . .	210 235
XVII, XVIII.	The determination of the influence of the eyeball on the growth of the orbit by experimental enucleation of one eye in young animals. (W. ERNEST THOMSON.) (Page 258) . .	260 261

LIST OF DIAGRAMS, ETC., IN TEXT.

FIG.		PAGE
1.	Visual field. (Rayner D. Batten)	73
2.	Visual field. (W. Lang)	99
3.	Visual field. (G. Anderson Critchett)	125
3 *a*.	,, ,, ,,	125
4.	Visual field. (J. Herbert Fisher)	128
5.	,, ,, ,,	128
6.	,, ,, ,,	130
6 *a*.	,, ,, ,,	130
7.	,, ,, ,,	133
7 *a*.	,, ,, ,,	133
8.	A model to assist in the teaching of ocular refraction. (H. Secker Walker)	142
9.	A capsulotomy knife. (Gustavus Hartridge)	143
10.	Recording colour perimeter. (J. H. Tomlinson)	145
11.	A portable refractometer. (C. Blair)	156
12.	New pattern of eye speculum. (H. E. Drake-Brockman)	162
13. 14.	Photographs of patient before and after the performance of a modified Hess's operation for ptosis. (G. W. Roll)	177
15.	The evolution of the colour-sense. (F. W. Edridge-Green)	186
16.	,, ,, ,, ,,	189
17.	The "amblyoscope." (C. Worth)	248
18. 19. 20.	Various object-slides used in the "amblyoscope." (C. Worth)	249
21. 22. 23.	,, ,, ,, ,,	251
24. 25.	To illustrate "So-called paresis of divergence." (G. A. Berry)	272
26. 27.	,, ,, ,, ,,	277
28.	Visual field. (A. Hugh Thompson)	287
29.	,, ,, ,,	287
30.	,, ,, ,,	289
31.	,, ,, ,,	289
32.	,, ,, ,,	290
33.	,, ,, ,,	290

REPORTS.

I. DISEASES OF THE CONJUNCTIVA.

1. *Tuberculosis of conjunctiva.*

By J. Herbert Fisher.

G. B—, æt. 18, milkman.

Family history.—Father and mother alive and well. Is one of a family of ten, three of whom have died. Two died in babyhood of convulsions, and the other said by mother to have died of "phthisis," which was the word on the death certificate. Father's family stated to be consumptive.

Previously patient's health has always been good.

Two months before I saw him first, on October 1st, 1900, the patient noticed the right side of face beginning to swell; a fortnight later he observed the upper lid of the right eye was commencing to swell; there was some yellow discharge from the eye. Three weeks after the swelling of face began he found glandular enlargement extending into the neck under the jaw.

When first seen the lids presented about the same appearance as at present; they were swollen and œdematous, with slight dusky discoloration of skin. The palpebral conjunctiva of both upper and lower lids was converted into an œdematous gelatinous-like tissue, the surface presenting a cauliflower-like appearance, due to flat-topped excrescences closely pressed together; no

ulceration or localised induration anywhere. It is quite unlike trachoma, and agrees more with the condition of spring catarrh. The ocular conjunctiva is free; there is some excess of mucoid secretion. The thickened heavy lids droop, and the eye can only be opened very imperfectly by the patient. The granulations do not bleed very easily.

In parotid region is a large glandular swelling extending to the submaxillary glands at the angle of the jaw on the right side. Glands are discrete and very hard. The skin over them is reddened and œdematous, and the swelling has the appearance of rapidly breaking down. Under hot lead lotion dressing the acute inflammation here has, however, subsided, leaving a mass of very hard discrete glands. When first admitted the temperature rose in the evening to 100° F.—102° F., but has not now been above 99° F. for one week.

There is nothing to suggest syphilis, either inherited or acquired.

The lungs show no signs of disease.

There is no general glandular enlargement, no lupus of skin.

The urine is normal.

Microscopic sections of the altered tissue of the palpebral conjunctiva show abundant multi-nucleated giant cells, with exudation of inflammatory leucocytes, and are reported on by Mr. Shattock as diagnostic of tuberculosis. Success has not as yet attended the search for tubercle bacilli in the sections.

(A microscopic section of the tissue was shown.)

(*Card specimen. October 18th*, 1900.)

Mr. Johnson Taylor suggested the use of lactic acid in increasing doses, beginning with 25 per cent., and going up to 90, as used in laryngeal phthisis. He thought that would be less destructive than other measures.

The President said that ethylate of sodium was

suggested some years ago by Mr. Brudenell Carter for such cases. It would have to be used with great care.

Mr. Fisher, in reply, thanked Mr. Taylor for his recommendation. He had himself thought of lactic acid, but did not know how the eye would tolerate it, or in what strength it should be used.

2. *Primary chancre of retro-tarsal fold of upper lid.*

By Arthur W. Ormond.

The patient presented himself at Mr. Higgens' out-patients', Guy's Hospital, on October 15th, 1900. There was noticed some drooping of the upper lid of the right eye, which was swollen and red. At the upper part a swelling was seen which on manipulation was found to be hard, rounded, and about the size of a bean, freely moveable on the deep structures, and not adherent to the skin. On everting the lid the surface of the conjunctiva was red and congested everywhere, but where it passed over the swelling was of a deeper colour. The swelling had a smooth surface, except in the centre, where there was a slight depression, with radiating striations coming from it. There were enlarged glands at the angle of the jaw.

He was seen again on October 29th, when a well-marked papular rash covered his trunk and thighs.

(*Card specimen. November 8th*, 1900.)

The President said it was an extremely interesting case of chancre, and the secondaries were well developed. There could be no doubt as to its nature. It was important to ascertain how it came about. When he was in the Black Country visiting one of the large iron

works, accompanied by a medical friend, he saw a workman get a foreign body into his eye, and before either of them could intervene a comrade had got his head between his knees, and was doing his best to extract the foreign body with a chisel; and then, rather to his disgust, he found that the man used nasal mucus afterwards, and his medical friend explained that this was the usual custom. That was their effort to replace the denuded corneal epithelium for the time being. Under those circumstances it was quite possible that the disease in the present case was conveyed in some such way. They had frequently seen a primary Hunterian chancre in the lid in infants, but that had been caused at birth *in transitu*. He had seen several cases, two or three when he was clinical assistant at Moorfields, and one at St. Mary's Hospital. Probably others present had seen cases, but not exactly in the situation.

Mr. Snell said he had seen about twenty cases of chancre about the eyelids, but the interest of the present case was the situation of the lesion. He had related to the Society a few years ago the case of a chancre which was inside the lachrymal sac. That, he believed, was the only case of the sort on record. The remark made by the President appealed to him, living, as he did, in Sheffield. At each of the large works there were men who had reputations for removing foreign bodies from the eye, and some of them removed thirty or forty a day. Some of the men were very dexterous in removing foreign bodies. He had not previously thought so much of the possibility of syphilis being conveyed in that way as of the probability that the efforts to remove the foreign bodies would occasion the abrasions of the cornea to become septic. No doubt many of them did. He had for the last year or two been endeavouring to get the men at these works to use spuds for the purpose instead of knives and pins, the spuds being made of iridium and platinum, so as to be readily sterilised in a flame.

Dr. Edridge-Green said with regard to the probable

method of infection, he had observed that at Hendon a common practice among the poor people was to remove a foreign body from the eye by means of the tongue.

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3. *Conjunctival growth (? pinguecula).*

By W. C. Rockliffe, M.D.

Florence C—, æt. 12, attended at the Hull Roya Infirmary, August, 1900.

History.—The mother states there was noticed at birth a "small reddish pimple" close to the cornea on the outer side of the left eye. This has gradually increased in size.

Present condition.—There is a crescentic pigmented thickening of the conjunctiva surrounding about one third of the cornea to the outer side of the left eye, with several small vessels and one principal conjunctival vessel running into its substance. The growth measures 1 cm. in length, 6 mm. at the widest part, and is about 2 mm. thick in the centre; it is entirely confined to, and freely movable with, the conjunctiva, except at the margin of the cornea, where it appears to be attached and to slightly overlap. The tumour does not appear to have enlarged during the last three months it has been under observation, but the parents think otherwise. The submaxillary glands are normal, but the cervical lymphatics are enlarged. There is a distinctly strumous family history. V. $\frac{6}{9}$ reads No. 1 J. F. of V. free. T. n. Pupils oval. F. N., dark choroid. The patient is very dark, has a mole on her cheek, and is of purely English parentage.

The scar on the growth is where a portion was removed on December 4th for examination by the *Clinical Research Association*. I regret as yet not to have received their report.

Dr. Rockliffe stated the section shown was one of the conjunctival tumour exhibited at the December meeting, when numerous diagnoses were made. The growth had been reported as a "squamous epithelioma." But the opinion of those who had examined the section that evening was so different that he ventured to ask that a special committee might be appointed to report on the growth. (This was agreed to.)

Report of Committee on Dr. Rockliffe's Growth of the Ocular Conjunctiva.

Two sections of the growth have been submitted to us stained with logwood and eosine. On one side of the sections is a thick layer of fibrinous material, thickest about the centre, immediately beneath which is a thin layer of stratified epithelium. Between this and the main mass of the growth is some dense fibrous tissue of variable thickness, in places the growth coming almost into contact with the stratified epithelium, whilst in others it is widely separated. The surface epithelium has processes projecting down from it into this fibrous tissue, and also, where the fibrous tissue is thin, into the growth itself. There are, besides, distinct islets of epithelium embedded in the substance of the growth.

The bulk of the growth is composed of round and oval cells of variable size, arranged in imperfect alveoli. The method of staining employed for the sections does not allow of our definitely stating whether strands of fibrous tissue separate each individual cell one from the other, or whether they lie directly in contact. There is a small amount of pigment scattered throughout the growth, contained for the most part in its cells.

In the bands of fibrous tissue dividing the growth up into imperfect alveoli, channels are seen having all the appearances of blood-vessels. In one part of the specimen there are some much larger channels lined by a single layer of endothelial cells, and which appear to be

dilated lymphatics. Some of these are devoid of contents, whilst others of similar appearance contain masses of cells like those of which the growth is composed.

We have compared the section of the growth with those of an undoubted mole of the skin, and find that in the character and arrangement of their cells they present many features in common. Indeed, we are inclined to regard the specimen as a congenital mole located in the conjunctiva.

E. Treacher Collins.
C. Devereux Marshall.

4. *Sequel to case of tuberculosis of conjunctiva, shown July 6th*, 1900. (' Trans. Ophth. Soc.,' vol. xx, p. 51.)

By Walter H. Jessop.

On July 13th, 1900, the patient was operated on under chloroform. The large ulcer with its hardened edges in lower conjunctival sulcus of left eye was dissected up with a scalpel and removed. The infiltration and thickening were found to extend to the periosteum at the outer side of the orbit; a considerable piece was dissected from the upper palpebral conjunctiva on the outer side, and from the bulbar conjunctiva as far as the cornea in the middle line. The growth invaded the cornea at the outer side, and the cornea there was scraped with a sharp spoon, as was also the base of ulcer near the periosteum; the palpebral conjunctiva of the lower lid was also scraped.

On August 3rd the eye had quite quieted down, but there was still some thickening at the canthus, which I thought might indicate a recurrence. There were also some small pale raised patches on the inferior palpebral conjunctiva.

On August 4th the patient went to Margate Sea-Bathing Infirmary, where he remained for three months.

On his return from Margate in November he looked in perfect health, the condition of the eye was most satisfactory, and the disease apparently eradicated and cured.

Left eye.—Palpebral aperture a little smaller than the right. At outer canthus is a thin cord-like symblepharon, and at its corneal end is a conical nebula corresponding to the part of the cornea scraped; in lower sulcus are some small adhesions. Cornea towards centre: a few small nebulæ.

The pre-auricular gland is no longer discharging, and the ulcerated surface has healed over.

(*Card specimen. January 31st*, 1901.)

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5. *New growth on conjunctiva and cornea.*

By W. Lang.

Annie H—, æt. 16.

Family history good.

Personal history.—Good health. First noticed a little swelling on left eye when about eight years old; seemed to be "like a cold at first," and has gradually increased since.

Clinical appearance.—Upon the lower part of the globe there is a small fleshy growth about 15 mm. in diameter and 3 mm. high, springing from the ocular conjunctiva and spreading over the lower third of the cornea. Its surface is evenly smooth, but it is divided into lobules or leaflets, which separate from one another on manipulating the growth through the lower lid. The upper margin of the corneal portion is sharply defined. Along its edge are several small white punctate opacities in the corneal substance. When looked at through a magnify-

ing lens the growth is seen to be supplied by vessels which branch greatly, but do not anastomose laterally, as if they were coursing in separate villi.

The growth was excised and examined with the microscope. It proved to be a papilloma covered with a thick layer of epithelium.

(*Card specimen. January 31st*, 1901.)

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6. *Notes of a case of membranous conjunctivitis.*

By J. F. Bullar.

The case occurred in an infant under the care of Dr. Whiteley, of Downton. I have put the facts in the order in which they happened, not as we found them out.

The child was born on the 28th of October last; both the mother and child, and everyone in the house were perfectly healthy.

Dr. Whiteley saw it every day until November 5th, when it was quite well.

On November 7th it was noticed that there was something wrong with the right eye. The nurse, an old country woman, said, "It is only a cold; I have cured lots like that; there is no need to tell the doctor," and with that she opened the child's eye and spat in it and wiped it with a general purposes cloth used in the bedroom.

The eye got worse, and on November 11th Dr. Whiteley was sent for. He found the lids much swollen and considerable chemosis.

On November 12th I saw the case with Dr. Whiteley.

The baby was apparently in perfectly good health; a fine baby, well nourished, bright, and taking the breast naturally. The left eye was natural and healthy. The

lids of the right eye were swollen a good deal, and the ocular conjunctiva very red and swollen. I could not see the whole of the cornea, but what I could see was clear.

Lining the lids there was a yellowish-white membrane not extending to the ocular conjunctiva. On taking hold of the membrane at the middle of the upper lid with forceps the whole membrane lining both lids came out very easily in one piece; it merely lay on the conjunctiva and was not attached to it. The palpebral conjunctiva beneath the membrane was red and bled very slightly. There was slight abrasion on the outer surface of the lids, also covered with the same kind of membrane, which peeled off very easily.

I recommended that the eye should be washed every hour, and a solution of nitrate of silver (two grains to the ounce) dropped in every two hours.

I did not see the child again, but heard from Dr. Whiteley that the swelling of the lids and chemosis subsided, and that there was a central ulcer in the cornea.

On November 17th a membrane appeared in the right side of the nose and fauces, and there was difficulty in sucking; the temperature was 100·4° F. The membrane then extended to the whole of the fauces, and on November 20th the child died in convulsions.

Dr. Whiteley tells me that there had not been any diphtheria in the neighbourhood before this case, but that during the baby's illness another child in the house had a white patch in the throat and a temperature of 104° F., but this child got quite well in forty-eight hours without any special treatment. Three or four months before the children in the house had measles, followed by nasal discharge lasting some time.

It is unfortunate that I did not preserve the membrane for examination, but when I saw the child it did not occur either to myself or to Dr. Whiteley that the condition was serious except in regard to the eye.

The healthy condition of the child and the easy

removal of the membrane led me to believe that the case was one of that type of membranous conjunctivitis which yields to simple local treatment. Of such cases I saw two or three at St. Bartholomew's Hospital, and I have since seen two at Southampton, but they are evidently not very common in that neighbourhood, for these are the only two among about 17,000 cases of which I have the notes.

I have never before seen a case which I had any reason to regard as diphtheritic from a clinical point of view.

I have reported this case to the Society in the hope that some light may be thrown on the subject by other members. At present it seems as difficult to define diphtheria of the conjunctiva as of the throat.

The PRESIDENT thought it would be well, in a case like that, to apply quinine locally. It could certainly do no harm, and would probably do great good. Mr. Tweedy was careful to stipulate that the quinine must be very pure. Mr. Tweedy and he attended a case of conjunctivitis in which they suggested the use of antitoxin, but public faith in it was not then so strong as now, and the offer was declined. They used quinine, and certainly the result was satisfactory. He thought it was not so contagious as general diphtheria.

Mr. SYDNEY STEPHENSON saw every year a fair number of cases of conjunctivitis associated with membrane, and his rule in hospital was that if he felt any doubt as to the nature of the condition, he ordered a liberal dose of antitoxin forthwith, without awaiting the results of a bacteriological examination. He had seen nothing but good result in such cases, and where he had neglected to use that remedy he had had reason to regret the omission. For example, a child was admitted to the North-Eastern Hospital for Children last year, but was not seen by him until the third day. The house surgeon had not used antitoxin, but had trusted to local treatment. He, Mr.

Stephenson, ordered it when he saw the case, but the child died twenty hours afterwards, the antitoxin having been evidently used too late. The diphtheritic process extended to the nose and throat in the same way as it appeared to have done in Dr. Bullar's case.

Mr. Devereux Marshall said he felt certain antitoxin was much neglected in those cases. Physicians who had used it greatly for throat cases said they never had any bad result from it. In the cases in which he had seen it used it had acted like a charm, and the membrane in the eye had disappeared as quickly as from the throat. Although he should still use quinine locally, yet he could not say he had seen such remarkable results from it as Mr. Tweedy had obtained. In such a case he, Mr. Marshall, would not hesitate to use antitoxin, and then verify the diagnosis afterwards bacteriologically.

In reply to the President, Dr. Bullar said the child's temperature was not taken, for it was not usual to do so in ordinary purulent conjunctivitis. The membrane coming away so easily was scarcely diagnostic of diphtheria.

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7. *Spring catarrh in the quiescent stage.*

By W. T. Holmes Spicer.

Colin M—, æt. 18, electrical engineer, was first seen on April 3rd, 1901. He has been living in Johannesberg, South Africa, seven years; during the hot season in the last three years has suffered from great redness and irritation in his eyes; he is an engineer in the mines, and attributed the irritation to the heat and dust in his work. During the cold season he has been free from irritation. He has had treatment of various kinds

for granular lids, and has undergone operations on his lids for the granulations without benefit. He came to this country for further advice, and during the voyage he experienced great heat while crossing the line, and had a more severe attack in his eyes than he has yet had. Since his arrival in this country he has had no trouble at all.

He has a father and mother, three brothers, and four sisters, all of whom have healthy eyes.

To a general inspection the eyes and lids appear normal, and free from any signs of congestion.

On everting the lids the milky appearance of the conjunctiva is most marked; the lower lids show slight papillary roughening of the conjunctiva. The conjunctiva of the upper lids is not congested, but is rather pale owing to the milky appearance; the tarsus and retrotarsal fold are covered all over with large pavement granulations having a flattened surface owing to pressure, and a roughly hexagonal outline from contact of the granulations with others immediately surrounding them; some of the granulations on the fornix are pedunculated. There is no trace of any formation on the limbus.

He was advised to remain in this country, as a return to Johannesberg would probably bring about an aggravation of his condition in spite of treatment, but this he was unable to do. (*May 2nd*, 1901.)

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8. *Further notes of a case of melanotic sarcoma of the conjunctiva.*

By J. B. Lawford.

At the meeting of this Society in June, 1896, I showed a patient, Mrs. R—, æt. 57, in whom the right ocular conjunctiva was the seat of comparatively recent but increas-

ing pigmentation. The notes of the case were published in the Society's *Transactions*, vol. xvi, p. 50.

The subsequent history of the patient, so far as it is known, is as follows:—In July, 1896, a flattened pinkish nodule appeared in the conjunctiva and subconjunctival tissue, close to the upper nasal border of the cornea, and soon after a second similar growth developed below the first. They were apparently unpigmented. The pigmented area increased in extent and became darker.

In August, 1896, the cornea became hazy and grey in its lower central part, the tension of the eye was slightly raised and some pain was experienced. No intra-ocular changes were detected.

In September, 1896, the patient consented to have an operation, and on October 1st the eyeball and the whole of the affected conjunctiva were removed.

The specimen was handed to Mr. Devereux Marshall (at that time Curator of the Museum at the Moorfields Hospital) who very kindly prepared sections of the eye and the conjunctival growth.

The pigmented portion of the growth consisted of small spindle-shaped cells, deeply pigmented, and situated in the conjunctiva and subconjunctival tissue. The pigmented cells were, in places, quite superficial; they were spreading into the epithelium over the margin of the cornea at one point.

The nodule of growth referred to consisted mainly of loose connective tissue, containing large blood-vessels. There was a small amount of cellular structure and very little pigment. No apparent invasion of the sclera. The intra-ocular structures showed no trace of new growth.

In May, 1897, the patient was reported to be very ill at her house, with headache, sickness, unconsciousness. The medical man in attendance sent to the hospital to inquire as to the nature of the eye disease, and evidently suspected intra-cranial tumour. These symptoms passed off and the patient recovered her usual health.

In October, 1897, patient was readmitted to hospital

with a hard pinkish nodule of new growth, "as large as a red currant," in the upper part of the orbit; some pigmentation at its base. The notes on admission state that her "general health is not good; the woman is thin and very sallow, but has no physical signs of metastatic growths."

October 21st, 1897, orbital growth removed.

Mr. Marshall kindly prepared sections of the growth, which proved to be a mixed round and spindle-celled sarcoma. The greater part of the growth was unpigmented; in one part a moderate amount of pigment was visible in the sections.

On August 11, 1898, patient came with a recurrence in the orbit; the nodule of new growth was extremely vascular in its anterior part, and there had been slight hæmorrhage from it on two occasions.

On September 1, 1898, the growth was thoroughly removed; it had no attachments to the wall of the orbit. The patient made a very slow recovery.

Dr. L. L. Jenner (then the superintendent of the Clinical Laboratory at St. Thomas's Hospital) reported, "The tumour is a sarcoma; the cells are chiefly spindle cells, and there is a considerable quantity of pigment."

In April, 1899, a pigmented spot about the size of a small pea was noticed on the inner surface of the lower lid, a situation in which there had been no growth previously.

On September 11th, after a temporary absence, patient returned with a recurrent growth in the upper part of the orbit. The small pigmented patch in the lower lid had increased to a slight extent.

No evidence could be obtained of secondary deposits in internal organs, but the cachectic appearance of the woman suggested systemic infection.

On September 14th, 1899, the entire contents of the orbit were removed. The patient made a good though slow recovery.

The growth was examined by Dr. Jenner, and reported to be a melanotic sarcoma.

Patient was seen for the last time on March 20th, 1900; at this date there was no sign of recurrence in the orbit. She had been ill with an attack of "influenza," and had had on one occasion severe epistaxis.

Patient died at her home on April 28th, 1900, after severe vomiting persisting for about thirteen hours. She had been out the previous day. No post-mortem examination was made; indeed, I did not hear of her death for some time after the event. (*July 5th*, 1901.)

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9. *Conjunctival bridges and pouches; union of retro-tarsal folds.*

By Major H. Herbert, I.M.S., Bombay.

The condition which I purpose to call, for want of a better term, a conjunctival bridge, is one of the rarer results of conjunctivitis, and appears to have hitherto escaped notice. Its nature will be readily understood from a glance at the accompanying sketches showing a number of everted upper lids, which, though mere outlines, are correct as regards proportion and actual measurements.

Case 1.—February 19th, 1898. A Parsee woman, æt. 35. A broad fold of conjunctiva came down from the right upper fornix to be attached to the tarsal membrane, about 4 mm. from the edge of the lid. There was no question of any line of attachment above; the fold simply widened out as it passed upwards into one of the normal retro-tarsal folds. A probe could be readily passed under either curved lateral margin, to be arrested at a point about 2 mm. from its outer margin. The curve of this outer border was a double one, owing to a second small

PLATE I.

Illustrates Major H. Herbert's paper on Conjunctival Bridges and Pouches (p. 16).

Case 1.

a. *b.* *c.*

Case 2.

Case 3.

Lett eye. Right eye.

Case 4.

Case 5.

Case 6.

Case 7.

Fig. 8.

Left eye (fold pulled down by probe).

Right eye.

Case 8.

To illustrate Major H. Herbert's communication on Conjunctival Bridges and Pouches (p. 16).

Adlard & Son, Imp.

union of the fold with the subjacent membrane. The tarsal conjunctiva was slightly rough and injected, but not scarred. The fold was divided near its tarsal attachment; and at the next inspection, a few days later, there was found a second smaller underlying fold, formerly concealed by the broader superficial fold. A probe passed under most of it, but was stopped near its outer border. Two days later still a third small band was found, which had lain beneath the two already divided. A probe passed completely beneath it at its upper part only. Whatever slight union there had been between the three folds of membrane, arresting the passage of a probe between them, seemed to have given way when the folds were cut.

Case 2.—April 2nd, 1900. A male Hindu, æt. 22. Had had both upper lids operated on formerly by a "hakim" for entropion by simple removal of pieces of skin. Eversion of the right upper lid revealed a single narrow fold near its outer end, attached to a strip of dense scar in the sulcus subtarsalis. A probe passed easily beneath the band. Elsewhere in the conjunctiva there was thin uniformly diffuse scarring, the result of trachoma.

Case 3.—October 25th, 1900. Male, æt. 20. "Bridges" in both upper lids. About the middle of the left lid there was a single narrow attachment a little below the upper border of tarsus; after its division a still smaller underlying one was revealed (about the point × in the figure). The right upper tarsus presented three separate attachments, all of the one retro-tarsal fold, and no underlying adhesions. The upper tarsal conjunctiva of both eyes was slightly rough and injected, with some traces of scarring; cicatrisation was also present in the lower fornices, with remains of confluent trachomatous lymphoid tissue.

Case 4.—November 12th, 1900. Male, over 50 years. A single attachment, 5 mm. broad, close to the upper margin of the right tarsus about its middle. The con-

junctiva of the upper lid was otherwise normal, except for slight injection; there were some faint scar lines in the lower fornix.

Case 5.—November 30th, 1900. A female child, æt. 2½—3 years. A single fold, 5 mm. broad, coming down over the right tarsus slightly towards its inner end, and united with it about halfway down. There were trachoma follicles on both lids of both eyes, with a line of scar forming in the right lower fornix; slight injection, rather more on the right side; no roughness.

Case 6.—December 11th, 1900. Hindu youth, æt. 17. Two conjunctival bridges, one broad and one narrow, with a minute hole in it, belonging to the one retro-tarsal fold, attached a little beyond the left upper tarsus. In ordinary eversion of the lid they were not seen; their exposure required full depression of the fornix. There was mild chronic trachoma in both conjunctivæ,—follicles with tarsal roughness and injection; scarring about the upper border of the left upper tarsus, and in the lower fornix; only traces of it on the right side.

Case 7.—December 20th, 1900. A Mussulman student, æt. 19. A broad fold, more in the outer part of the left upper lid, with a somewhat oblique attachment to tarsal conjunctiva, descending lower at its outer end. On the right side there was an even broader fold, occupying two thirds of the length of the lid, but it was attached mainly above the tarsus, coming down over it only at its outer end, and thus it nearly escaped notice altogether. There were some patches of scar over both upper tarsi, and rather more in the lower fornices and on the lower lids; in addition only slight injection and very slight roughness.

In addition to these nine upper lids of seven patients I have noted two instances of small inconspicuous bridges in the lower conjunctiva, redeemed from insignificance only by

their relationship with the more striking conditions just described. One, in a man, was between two folds of the lower fornix of a conjunctiva affected by chronic trachoma with moderate scarring. In the other case, a woman, a fold from the fornix was attached to the conjunctiva just below the tarsus; a small line of scar ran outwards in the membrane from this point.

The greater frequency and extent of the adhesions found in the upper lid is evidently related to the much greater expanse of the upper reflexion of conjunctiva, from which, during inflammatory attacks, swollen folds must descend as a tongue of tissue between lid and eyeball. The permanent adhesions formed with overlying tarsal membrane have in some cases been multiple along the summit of one fold, in others multiple through the attachment of two or even three folds. In no case was there a history of injury.

I have only seen two cases of conjunctivitis with such union of folds actually taking place. In one instance the surfaces of three folds of conjunctiva in the lower fornix were glued together by membranous exudation, and bled when separated. The other case was more interesting.

January 1st, 1901.—A Hindu youth, 18—20 years. Considerable inflammation of both conjunctivæ apparently subsiding. Dusky œdematous swelling of the lids, membranous non-adherent discharge, considerable ocular as well as palpebral injection, palpebral membrane swollen but smooth. In the right lower fornix there was a band of tough adherent membrane apparently becoming organised, binding together the summits of two folds, which were otherwise free or only slightly adherent to one another. In the left lower fornix there were two such bands of membrane. Near the outer end of each upper lid, when completely everted, was seen a portion of a retro-tarsal fold drawn down and united to the conjunctiva just above the tarsus by means of tough membrane. A probe passed easily beneath each fold. None of these "bridges" or adherent folds would have been noticed had not special attention been directed to them.

We have no means of judging whether all of the old permanent adhesions above described had been formed in this way through the instrumentality of fibrinous exudation. The large proportion of the cases showing clear signs of trachoma, past or present, is noteworthy; also the early age of one of the patients. The number of the above cases, which were collected within a few months, shows that in this country they are not extremely rare. They are very easily overlooked, even when the displaced folds come down over the tarsus, since the appearance of the folds may be quite similar to that of the tarsal membranes over which they lie, and when they are situated entirely beyond the tarsus they may, of course, still more readily escape notice. And since the adhesions give rise to no definite symptoms beyond, perhaps, tending to keep up a slight conjunctival hyperæmia by dragging on the lid, one can only expect them to be discovered, as a rule, by accident. On the other hand, they must be very rare in Europe, or one would have heard something of them before now. The conjunctivitis of this country is remarkable, not only in including much trachoma, but also in furnishing a large number of *untreated* and *ill-treated* inflammations; possibly here lies the explanation of these curious sequelæ.

Another question arises thus. In a boy seen on October 23rd, 1900, there was what appeared to be a broad conjunctival bridge on the left upper lid (see fig. 8). A probe, however, passed under it only a very short distance. The fold had adhered not only at its summit, as in the above-described cases, but almost completely over its surface also, leaving only a shallow pouch or pocket under each lateral margin.* It is obvious that, had the union been only a little more complete, the fold would have entirely disappeared, and no indication would have remained of the

* Underlying the inner margin of the fold and extending up into the fornix was a rather ill-defined cyst, with turbid fluid contents; evidently the obliteration of underlying epithelial surfaces had not been complete. The condition is suggestive as to the formation of the cysts not uncommonly found in the fornices together with scarring.

mode in which much surface had been lost to the retro-tarsal membrane. There was a good deal of cicatricial tissue in both upper and lower fornices, with remains of a trachomatous lymphoid ridge in the lower, and the case would have been classed simply as retraction of fornices from scarring. The question arises whether retraction of fornix is only rarely, or perhaps fairly commonly, due in part to such union and obliteration of retro-tarsal folds.

P.S.—Since the above was written, a systematic search among out-patients has unearthed several additional instances of similar conditions, chiefly retro-tarsal pouches, associated in most cases with definite cicatricial evidence of past trachoma.

1. February 16th, 1901.—A Jewess, of over 20 years. A narrow, tube-like pouch, 5 mm. long, lying just above the left tarsus. It extended outwards from the middle of the lid, and its mouth opened outwards. Towards the outer end of the right upper lid, above the tarsus, was a small shallow pocket directed upwards. In both conjunctivæ there was a good deal of scar tissue, diffuse beyond the tarsi, reticulate over the upper tarsi.

2. March 9th.—A male Hindu, 20—25 years. A small pouch opening outwards, only 2 or 3 mm. deep, above the left upper tarsus, outside its middle. At the outer end of the right upper lid, above the tarsus, was a pouch 3 or 4 mm. in depth, of which the mouth was directed inwards *towards the centre of the lid*, thus differing from all others that I have seen. Scarring diffuse except over upper tarsi, where islands of rough red membrane were left. The outer part of the right upper tarsal conjunctiva, corresponding exactly with the pouch above, was scarred; whereas just within this it was free from scar. Corneæ opaque from pannus and from ulcers.

3. March 11th.—Male Hindu, æt. 16. An imperfect "bridge," nearly 5 mm. broad, just above the left upper tarsus, about its middle. A probe was passed readily under either end, but was arrested by complete union under the middle of the band of tissue. Cicatricial tissue disposed rather in a network over both upper tarsi, more diffuse elsewhere; with some trachomatous, confluent lymphoid tissue remaining towards the outer ends of lower fornices and lower lids.

4. March 12th.—A Mussulman girl of 6 years. A very small bridge reaching down on to the left upper tarsus, near its outer end. About the upper border of the right upper tarsus, situated rather towards its outer end, and opening outwards, was a pocket or pouch, 6 mm. long. Diffuse scarring beyond tarsi, upper and lower, with a network covering upper tarsi.

5. March 12th.—Male Hindu, æt. 23. Shallow pockets over outer parts of both upper tarsi, opening outwards. Diffuse thin scar tissue everywhere, thicker lines in each sulcus subtarsalis, slight entropion.

6. March 15th.—Male Hindu, 30—35 years. A fine white line, extending throughout the middle third of the left lower fornix, showed the union of two folds of conjunctiva, complete outwards, but affecting only the summits of the folds for the inner 4 mm. of the line, and thus admitting a probe under it into a narrow tube or pouch, the mouth of which was directed inwards. Very little cicatricial tissue elsewhere; a little beyond the left upper tarsus, and some doubtful signs in the right lower fornix.

7. March 22nd.—A native Christian, æt. 36. A narrow tube in the left lower fornix, almost 1 cm. long, extending outwards from the middle of the lid and opening outwards. There was a small shallow pocket also, just below the right

lower tarsus. There were some lines of scar in both upper and lower fornices, and some traces over the right upper tarsus. (*July 5th*, 1901.)

Mr. N. B. Harman said he saw such a case in a Boer prisoner, an Irishman. He had no history of eye disease, except of grit in his eye. The band was a very short, tight one, and when seen the eye was in a state of irritation. He had not an opportunity of treating it.

II. DISEASES OF THE CORNEA.

1. *Epithelioma of cornea.*

By Simeon Snell.

(With pathological report by E. Treacher Collins.)

Joseph W—, a royal reservist, æt. 45, was admitted under my care at the Royal Infirmary, Sheffield, on August 28th, 1900, in consequence of a swelling in front of his right eye.

Seven years previously he was struck by a piece of coal on the right eye, at the lower and inner part of the corneo-scleral margin. At this time the patient was working as a yard labourer. He noticed nothing amiss with the eye until three years later, when he saw a pimple on the spot where the eye had previously been struck. The growth very slowly increased in size. By May, 1898, it had become as large as a pea, covered over half the pupil, and interfered considerably with sight. He then went to the Wolverhampton Eye Hospital, where the growth was removed. It recurred, and the patient was under treatment at the Royal Hospital, Sheffield, in December, 1899. This present year it has increased much more rapidly, especially during the last few weeks.

Previous illnesses.—Syphilis twenty-two years ago. Has tertiary "reminders" now.

Family history.—Good.

Before joining the reserve he had been in the Royal Artillery. When a reservist he has occasionally worked as a yard labourer, and it was whilst thus employed that he received the injury mentioned.

Present condition.—He is a strong, healthy-looking man. Has not lost flesh. No cachexia. Right eyelids are

swollen. The whole anterior part of the globe is apparently involved in a new growth, which fills the interpalpebral space and widely separates the eyelids. On a closer examination it is observed to be more particularly associated with the inner part of the globe, and a small portion of the cornea is left uncovered externally through which the pupil is just discernible. On turning the eye very much inwards the patient is able to count fingers. The growth measures about one inch in diameter, and is about the size of a flattened walnut. The surface is red, irregular, and nodulated, and there is a slight discharge of pus, but no distinct ulceration.

The preauricular gland of the right side is enlarged to the size of a marble, is moveable, and the skin is not adherent. A few glands can be felt under the right jaw and one under the left.

On September 8th ether was administered, and the right eyeball was enucleated, the conjunctiva being also freely removed in the neighbourhood of the growth. Examination showed the tumour to be attached to the cornea at its scleral junction, and at its lower and inner margin. From this attachment it spread, mushroom-like, over the front of the globe, and covered the cornea.

On September 14th the patient was again put under ether with a view to the removal of the indurated glands. The preauricular gland was excised and found suppurating and encapsuled. It was thought not to be malignant, and so the removal of the glands in the neck was not performed. (*Card specimen. November 8th,* 1900.)

Report on the pathological examination of an eyeball received from Mr. Simeon Snell, September, 1900.

A right eyeball with a growth attached to it anteriorly. This growth extends over the whole of the front of the cornea, with the exception of about one third on the outer side; it also covers the front of the sclerotic as far as

the insertion of the recti muscles, downwards and inwards; upwards it does not extend much beyond the margin of the cornea. On antero-posterior horizontal section of the eyeball and growth it is seen that the latter does not extend into the interior of the globe. The cornea and sclerotic, where the base of the growth is attached, are thinned to about half their normal substance. The base of the growth at its point of attachment to the globe measures 13 mm. across; from there it fungates out, measuring at its widest part 17 mm. It is raised above the surface of the globe 8 mm. The structures in the interior of the eyeball appear healthy, except for one small oval patch of choroidal atrophy surrounded by pigment in the outer half.

Microscopical examination of sections of the front of the eyeball and growth show the latter to be an epithelioma. It is composed of irregular clumps of closely packed epithelial cells, with bands of fibrous tissue, containing blood-vessels, between them; also in places diffuse collections of small round-cells. In some of the clumps nest-like arrangements of the epithelial cells are to be seen, but in none of these are the cells very much flattened-out or cornified. In parts there are some well-formed prickle-cells. The growth has to a certain extent invaded the sclero-corneal tissue; beneath its base the laminated fibrous tissue is definitely thinned; there is there no trace of Bowman's membrane. Around the base of the epithelial processes, which are extending into the fibrous tissue, is a small amount of round-celled infiltration. There is also considerable round-celled infiltration along the track of one of the anterior ciliary arteries.

E. Treacher Collins.

2. *Growth on cornea.*

By A. Stanford Morton.

Albert L—, æt. 12, first noticed in May last a spot the size of a pin's head at the upper margin of the right cornea. When first seen at Moorfields on August 7th there was a yellowish circular patch about 4 mm. in diameter, not raised above the surface of the cornea, but extending in its substance to just beyond the upper margin of the pupil. There was slight swelling of the limbus and a conjunctival vessel ran over the surface of the mass, but there was no sign of inflammation. An attempt was made to shave off a portion of the mass, but, this having failed, a portion of the cornea was removed by transfixion, and the knife then entered a space occupied by yellow material, which was removed partly by scraping and partly by dissection. This was examined by the curator, Mr. Lister, who reports as follows:—Sections were made through the slice of cornea removed, which microscopically is found to consist (*a*) of the superficial layers of the cornea (epithelium, Bowman's membrane, and a considerable thickness of substantia propria), which are perfectly normal in appearance, on the posterior surface of which is (*b*) a thin layer of highly cellular tissue of new formation. This cellular tissue corresponds in position with the yellow substance seen in the substance of the cornea. It is composed chiefly of cells, with a small amount of fibrillar and granular matrix which contains a few blood-vessels. The cells have no definite arrangement, and their nuclei vary in shape and size (round, oval, or linear), while some are misshapen and degenerated. There are a few larger cells closely resembling those of the corneal epithelium, and also a few giant-cells, but no typical arrangement suggestive of tubercle. There is no appearance of any fat-cells, nor did scrapings of the patch stain

with osmic acid. With so small and incomplete a portion of the growth it is difficult to give any definite opinion as to its nature, nor is it possible to say with certainty whether the layer of new tissue was entirely enclosed between layers of the substantia propria, or whether it was continuous at any part with the surface.

(*Card specimen. December 13th*, 1900.)

The PRESIDENT said that in a similar case Sir William Bowman suggested that if the growth progressed and nothing else seemed to check it, a small incision or furrow should be made in the cornea in advance of it, so as to introduce a stronger cicatricial element in the hope of arresting its growth. He believed the procedure was not carried out. Perhaps Mr. Morton would consider the advisability of trying that plan.

Mr. H. GOLDSMITH said he had at present under care a boy, æt. 7 or 8, who had a slightly raised new growth on the corneo-scleral margin which was encroaching on the cornea. It seemed to be somewhat similar to Mr. Morton's case. There were two or three large vessels running to the margin of the growth, and the surface was covered with smaller vessels, giving it an opalescent pink tinge, but quite unlike that of interstitial keratitis. There was no staining with fluoresceïn. He had only seen it two months, and could not say whether it was progressing. There was a good deal of conjunctivitis of the upper lid on that side. Six weeks later the growth was advancing over the cornea, its lower edge being roll-like and capable of a slight eversion.

3. *Epibulbar sarcoma.*

By J. Herbert Fisher.

Patient is a man, æt. 28, invalided home from the army in India on account of the condition of the right eye. In May, 1899, at the end of a march, he noticed a little red spot on the white of the right eye; it gradually grew and was entirely removed by an army doctor in February, 1900; recurred, and a second operation was performed in May of this year; again growing, a third operation was performed in August of this year. The last operation only was under general anæsthesia, and he does not think either of the last two operations completely removed the growth, though the first he considers did do so.

There is a history of venereal disease once in 1895—a small chancre which lasted three weeks and was followed by no sequelæ.

He has had fever once in India (malarial).

His father's brothers and his own elder brothers died of consumption with coughs and blood-spitting.

Nine brothers and sisters, born after the patient, died when between one and five years old.

Patient does not appear to be the subject of congenital syphilis.

There is a firm, flat, yellowish-brown, cake-like mass on and involving the inner part of the sclera of the right eye; it is smooth on the surface and very vascularised; the conjunctiva seems adherent on its surface, and implicated. It overlaps and invades a segment of the cornea amounting to about one fifth of the corneal area; it is limited towards the centre of cornea by a sharp and almost vertical edge; the rest of the cornea is absolutely clear and uninvaded by any inflammatory infiltration. The pupil being uncovered the eye has vision $\frac{6}{9}$; there are no changes to be seen in fundus or media with a dilated pupil. Inwards, the new tissue implicates the plica semilunaris and to some extent the caruncle. It extends upwards and downwards well

beyond the ciliary margins of the lids, and appears everywhere fairly sharply limited. Movement is limited considerably in the outward direction, and to a slight extent upwards, giving rise to diplopia when the eyes are turned to an object up or to the right. The growth is vascular but quite unpigmented. There is no glandular enlargement to be felt. The left eye is normal.

(*Card specimen. December 13th*, 1900.)

I excised a small portion of the growth for microscopical examination, which confirmed the diagnosis. On December 19th, 1900, I removed the lids, globe, and orbital contents in one mass.

He is an example of what may be done by Thiersch grafting in cases of evisceration of orbit.

On January 2nd, 1901, a fortnight after the exenteration, I completely covered the granulating walls of the orbit with Thiersch grafts taken from the patient's arm. It was dressed at the end of a week, and the patient was able to leave the hospital a fortnight after the grafting with a skin-lined orbit. The only point where a graft failed was over the site of the lacrymal groove, and here the granulations are still exposed, though slowly being covered with skin; a graft which I put on this site about a month ago only took to a very limited extent. The advantage to the patient from this ready method of shortening the healing process is obvious. At the time that I grafted the orbit with Thiersch's grafts I did not know whether this had previously been done. I have since found references to three other cases by Continental surgeons; two of these put the grafts at once on the orbital wall at the time of evisceration, and the other waited for granulation, as I did. All three were successful.

(*Card specimen. May 2nd*, 1901.)

4. *Spreading opacity of the cornea following herpes frontalis.*

By W. T. Lister.

E. M—, æt. 58, married.

In July, 1899, she had an attack of herpes frontalis on the left side, from which there are a few scars on forehead still visible.

Six or eight weeks after this attack the left eye became inflamed, and she was treated for several weeks for corneal ulcer.

February, 1900.—I saw her at the Central London Ophthalmic Hospital for the first time. She then had a nearly circular opacity on the outer side of left cornea, which extended from the margin to one third across the cornea. She thought the spot was increasing in size, but had little or no pain. Treatment: dilute mercury ointment.

June, 1900.—The opacity had distinctly increased in size. Bordering the opaque area noted before was a band of hazy grey opacity about 2 mm. in breadth, and the edge of this was halfway across cornea. Her vision then was—R. $\frac{6}{6}$, L. $\frac{6}{6}$; fundi normal.

January, 1901.—The opacity is now slightly larger than it was in June, and the grey band has become distinctly more opaque.

On no occasion has there been any sign of inflammation, and the epithelium on the surface has always been smooth and bright.

Sensation of both corneæ diminished; left slightly less sensitive than right.

(*Card specimen. January 31st*, 1901.)

The President thought it might be well to try the effect of small doses of iodide of potassium.

Mr. Hartridge asked if there was ulceration at any

time, and if any attempt had been made to stain the patch with fluoresceïn.

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5. *Double symmetrical opacities of cornea (rare form) removed by operation.*

By P. H. Mules, M.D.

This case differs from any that I can find recorded, both in the position of the opacity and the means taken for its removal.

The patient, a German merchant æt. 70, closely engaged in business, had the following history. General condition that of perfect health; habits regular and abstemious. Eight years before operation had presented himself to me with small opacities at the inner margins of both corneæ. They were symmetrical, curved, and greyish brown in colour; they caused no inconvenience, and only extended a third of the distance between the corneal edge and the pupillary margin. Gradually the opacity drew towards the pupil, and after eight years presented the appearance of a rounded cone, chocolate-brown in colour, the apex being at the centre of the pupil, the base lying on the corneal edge and occupying a fifth of the cornea. Across the middle of each opacity was a fine crack through which was a clear reflex, showing an interruption of the opacity.

The opacity was considered to be a deposit of earthy salts beneath the corneal epithelium, the outcome of partial stasis of the corneal fluid, and removal by scraping was suggested and agreed to, as the opacities now seriously interfered with vision. On attempting removal by this means the opacities were found to lie in the layers of the cornea (interstitial), an appreciable thickness of corneal tissue covering them. They could not be scraped off, and only after prolonged manipulation extending over half an

hour could the pupil be cleared. This difficulty was due to the corneal tissue becoming opaque as the deposit beneath it was being picked off. No reaction followed, and the patient was satisfied to have the fellow opacity removed.

This time a different course was pursued. Instead of scraping, a thin sclerotomy knife was passed under the opacity, through the layers of the cornea, entering exactly at the upper margin of the opacity and coming out exactly at the lower; by a sawing movement the band was separated intact to the edge of the cornea, the knife edge reversed and carried to the apex, when the entire opacity was lifted off, with the thin layer of cornea in front of it, the whole operation not taking more than fifteen seconds. As before, no reaction followed, the cornea remaining perfectly clear. After a year there is no fresh deposit, the cornea is faceted somewhat, but the patient reads and writes with ease. (*June 13th*, 1901.)

The PRESIDENT said he had an absolutely similar case under care at the present time, which he was seeing at intervals of six months. The patient was a lady whose opacities were dark and earthy-looking, and were slowly encroaching towards the pupil. He had watched them for seven years, and they had not yet impaired the sight, therefore he had not felt it necessary to deal with them. The only difference between the case and Dr. Mules' was, that there was not any clear cornea showing through. He had long arrived at the conclusion that they were deeply seated in the cornea, and that no scraping would be of use. If he had to deal with them he would certainly follow the plan mentioned by Dr. Mules.

Dr. MULES said, in reference to his case, two cracks were visible, but he did not know how long the second one existed. Mr. Power had seen one but not the other. Mr. Nettleship said he had seen a case in which the cracks resembled those in crackled china, the cracks not running perfectly vertically. He thought the idea that it was

a slow stasis must be the correct one, otherwise he could not have got his sclerotomy knife so perfectly underneath it. It looked as if the layers had been separated somewhat.

III. DISEASES OF THE IRIS AND CILIARY BODY.

1. *Serous iritis with nodular formations in the anterior chamber.*

By Rayner D. Batten.

Mabel K—, æt. 14 years, has been under constant observation since March 8th, 1900, with well-marked bilateral serous iritis; gross keratitis punctata; deep congestion; posterior synechiæ. No gross change detected, but details of fundi dimly seen owing to general haze of lens. No vitreous opacities detected.

January 31st, 1901.—A nodule first appeared in the right eye (she had been seen two weeks previously, when nothing unusual was noticed). At the lower part of the anterior chamber and pressing back the iris was an irregular nodulated heap, about 1·5 mm. high, pink in colour. No vessels were detected in the growth at this time.

There are now (May 2nd, 1901) three separate deposits or nodules, all in the lower third of the cornea; the largest one at the lowest part of the anterior chamber, and one on each side of it. The nodules have not increased much in size since they were first observed, and are somewhat less nodulated. There is a well-marked vessel on the temporal nodule, and some finer ones on the inferior nodule.

No evidence of congenital syphilis, rheumatism, or tubercle.

When first seen she had a rapid pulse 112, and slight enlargement of the thyroid. This disappeared after a time, but later the thyroid again enlarged somewhat.

(*Card specimen. May 2nd*, 1901.)

Postscript.—When last seen (August 8th, 1901) the nodules had disappeared. The serous iritis persisted in both eyes.

2. *Serous iritis with persistent tachycardia.*

By Rayner D. Batten.

Amy E—, æt. 16 years.

Seen first in September, 1899, with well-marked serous iritis; gross keratitis punctata of the large irregular "cold-fat-drop" variety.

Pupils nearly fixed by posterior synechiæ: scarcely any dilatation under atropine. The condition has persisted since that date, varying considerably in degree, and is at present less marked than it has been previously. At one time there was abundant deposits on the anterior surface of the lens, also a form of keratitis, apparently interstitial, appeared in the centre of the left cornea, and some traces of this remain.

Whilst appearing in fairly good health she has throughout had marked and persistent tachycardia, the pulse rate varying from 104 to 140.

The cardiac impulse was to be felt outside the nipple line and to the right of the sternum. No cardiac murmur detected. She had some fine tremors of the hands, but no enlargement of the thyroid.

Dr. Guthrie, who has also seen the case, considers it an example of Graves's disease, and that there is probably an adherent pericardium.

The President knew of two cases of serous iritis associated with Graves's disease which he had seen during the last few years.

(*Card specimen. May 2nd*, 1901.)

IV. SYMPATHETIC DISEASE.

Wound of eyeball with prolapse of iris; enucleation of eyeball twenty days after the accident, followed fifty-four days later by sympathetic disease of the other eye; recovery.

By Sydney Stephenson, M.B.

History.—James S—, æt. $7\frac{2}{12}$ years, whilst playing with his sister, had a dirty steel nib accidentally thrust into his left eye. He was taken to the Evelina Hospital next day, December 31st, 1899. There was a family and personal history of tubercle, but none whatever of syphilis.

Upon admission the iris was found to be prolapsed through a small incised wound on the outer part of the ciliary region of the left eye. Under chloroform the piece of protruding iris was cut away, all attempts to reduce the same having failed. The eye was kept covered with a pad and bandage, and atropine drops (gr. ij) were applied thrice a day.

Progress.—For some days the wounded eye seemed to be doing well; but then the tension fell, the globe became markedly congested, and an exudation into the vitreous could be perceived through the transparent crystalline lens. On January 19th, 1900, twenty days after the accident, the condition of the wounded eye was as follows:—Eye irritable, deeply congested, and watery, but apparently painless. No perception of light; tension — 2. Wound in

ciliary region healed, but iris incarcerated and drawn towards the cicatrix; a few posterior synechiæ present; root of iris retracted; lens transparent; a deep, yellowish reflex can be recognised through the pupil. The right eye showed neither punctate keratitis nor optic neuritis. Under A.C.E. the affected eye was enucleated on the morning of January 19th. The child made an uneventful recovery, and was discharged from hospital on January 27th, 1900, after a stay of twenty-eight days.

March 16th, 1900.—The mother attended, and stated that two days ago (March 14th) she noticed the lad's right eye to be "a little pinkish," but, according to her statement, the eye did not water, dread the light, or seem to cause any pain. On examination there was a trifling ciliary blush, and after atropine had been applied, posterior synechiæ were found, distorting the regular outline of the pupil. Two or three spots of punctate keratitis were present at the lower part of the cornea. Tension was normal. The retinal vessels were tortuous, and the inner side of the optic disc was hazy. A diagnosis of mild sympathetic irido-cyclitis was entered. The eye was protected from light with a pad and bandage; atropine drops were ordered thrice a day; and a drachm of Mist. Hydrarg.* was prescribed three times daily. A note was made of the fact that there was no inconsistency between the objective signs of inflammation, on the one hand, and the statement that the eye had been inflamed for two days, on the other.

23rd.—No redness of the eye since last visit. Tension normal. Neither vitreous opacities nor definite papillitis is present. Pupil well dilated. Two small black spots on the upper third of the lens capsule represent posterior synechiæ, the capsular attachment of which has been ruptured by the atropine.

30th.—Vision (uncorrected) $\frac{5}{24}$. Tension normal.

* Each drachm of the *Mist. Hydrargyri* of the Evelina Hospital Pharmacopœia contains, perchloride of mercury $\frac{1}{6\frac{1}{4}}$th grain, and iodide of potassium 1 grain.

April 6th.—Pupil regular and widely dilated. No redness of the eye. No punctate keratitis. V. $\frac{5}{60}$ + 1·0 + 0·5 90° = $\frac{5}{9}$.

20th.—V. (corrected) $\frac{5}{6}$.

May 11th.—The gums, although not sore, have become spongy.

June 29th.—The gums are very spongy, the breath offensive, and some teeth are loose, but there is no purging or other abdominal symptom of mercurialism. The mercury and the atropine were discontinued.

Result.—The patient was last seen on May 14th, 1901—that is, sixteen and a half months after the accident,—when the condition was as follows:—Right eye: no external changes; V. $\frac{6}{6}$ and No. 1 Jaeger. T. n. Pupil 3·5 mm., readily responsive to light, to convergence, and to atropine. No synechiæ, punctate keratitis, or vitreous opacities. Fundus, except for tortuous retinal vessels, normal. Field of vision for white has its full peripheral extent. Left eye: socket white and free from secretion.

Pathological examination.—The enucleated eye was hardened for twenty-four hours in formalin diluted with nine volumes of water, and was then frozen and bisected. One half of the globe was mounted as a jelly preparation, while the other was embedded in celloidin and cut for the microscope.

The jelly specimen showed that a black line of incarcerated iris passed through the sclera somewhat to the outer side of the limbus. A greyish-white exudation lay in the fore part of the vitreous humour, and filled, perhaps, one fourth of the chamber. The rest of the eyeball appeared to be normal.

Microscopical sections were stained in various ways. The essential features of the following description were common to all the specimens examined. At the extreme periphery of the anterior chamber on one side there was an incarceration of the iris, which was covered with œdematous conjunctiva and subconjunctival tissue. Many particles of

pigment were present in the wound-canal, and some had been carried a short distance into the neighbouring tissues. The cornea showed no obvious changes. The iris was hypernucleated, and in the vicinity of the pupil rather wide adhesions existed between its uveal layer and the anterior capsule of the lens. Its vessels were distended with red blood-corpuscles. That the ciliary body was inflamed was shown by (*a*) its dilated blood-vessels; (*b*) its cellular condition; (*c*) hyperplasia of its uveal layer with extension of pigmented particles into its substance; and (*d*) an exudation from its inner surface, including tubular processes and numerous particles of pigment. This exudation was continuous behind the lens with a similar exudation from the ciliary body of the other side. The exudation ran into an effusion of pus-cells in the vitreous chamber, continuous with the thickened and inflamed retina. The peripheral parts of the lens were vacuolated. Sections were stained for bacteria, but with negative results.

(*July 5th*, 1901.)

The PRESIDENT thought it would be interesting to know whether there was any tenderness in the orbit of the enucleated eye.

Mr. STEPHENSON said he did not look for the point.

The PRESIDENT said he asked because his attention was directed to it by Professor Liebrich, who suggested ascertaining whether rather firm pressure in the region of the optic nerve in the socket of the other eye gave the patient pain.

Mr. MARSHALL asked whether fifty-four days was a record time. He had seen a case in which it occurred at from fourteen to twenty-one days, but never at so long an interval as in the present case.

Mr. SNELL said he had only met with two cases in which ophthalmitis had occurred after enucleation, but his recollection was that the period had been more like that mentioned by Mr. Marshall, but he had had several cases in which sympathetic ophthalmitis of a mild character had

taken place, and in which he had not felt he could enucleate the other eye. This was a question very difficult to decide, because it might turn out that the removed eye would in all probability have been the better of the two. Last winter he had a boy who had three relapses of sympathetic ophthalmitis; the first eye was not removed, and now he had both eyes in good condition. He did not think the point was sufficiently insisted on, that one might remove what might ultimately turn out to be the better eye. He did not know any case in which there was more difficulty in deciding than where the injury left a fair amount of sight and yet sympathetic ophthalmitis had arisen in the other eye. In several cases he had left the eye, and he thought that, on the whole, he had been satisfied with the results; and it was well recognised that the removal of the injured eye did not always stay the progress of sympathetic ophthalmitis when once it was started in the other. The case which specially drew his attention to the point he had mentioned occurred fifteen or more years ago. He declined to remove an eye, the patient was taken elsewhere, and the eye was enucleated, with disastrous results, as the sympathising eye was absolutely lost. He did not know what the result would have been if the sympathising eye had been left.

Mr. Stephenson, in reply, said the indication for the removal of the eye in his case was clear: the eye was blind, and there was pus in the vitreous chamber. He brought the case forward because it belonged to a class about which there was still much to learn. He would hesitate to say that fifty-four days was a "record" period, but it was much longer than in the majority of reported cases. The great difficulty was to distinguish between genuine and non-genuine cases. In the present case the good eye was examined before the patient left the hospital; therefore there was positive evidence that sympathetic ophthalmitis had not at that time commenced. He thought that the case bore out the point that, if sympathetic ophthalmitis came on after enucleation, it was likely to be of mild type. The

Committee of the Society (vol. vi, 1886) collected thirty authentic cases of the kind. In eighteen of them recovery occurred, in three the recovery was more or less incomplete, and in nine the eye was lost. That furnished, he thought, an instructive contrast with the results of cases of ordinary sympathetic ophthalmitis.

V. DISEASES OF THE CRYSTALLINE LENS.

1. *Ætiology of lamellar cataract.*

By Norman G. Bennett (introduced).

Considerable diversity of opinion exists as to the date of incidence, the exact causation, and the nature of the lenticular lesion which is characteristic of lamellar or zonular cataract. The object of this paper is to endeavour to throw some light on the matter chiefly by a consideration of those dental lesions which are frequently associated with the disease.

Mr. Treacher Collins, in his lecture at the Royal College of Surgeons in 1894 (1), gives a *résumé* of previous investigations into the histology of the disease, and a description of seven lenses which he had himself examined. Three changes were described corresponding to the striæ, the haze, and the white dots which are observed clinically, namely, fissures between the lens fibres at the boundary of the nucleus and concentric with it, small vacuoles in large numbers, round, oval, or elongated in shape, and larger spaces of irregular outline. A granular material appeared to occupy these large spaces, and was also seen in the fissured spaces; the small vacuoles contained a hyaline substance. These changes were not necessarily coincident in the same lens. In the majority of specimens small vacuoles occurred in the nucleus, usually more frequently towards the periphery, but the cortex was in all cases normal.

Various views have been expressed as to whether these changes are produced before birth or after, and whether the appearances described are the result of noxious influence during the development of the particular zone affected, or of a degenerative process occurring later. Schreiner believes that the vacuoles are due to the former

cause, and the concentric spaces to the contraction of the nucleus already formed.

Bernard Dub (2), arguing in favour of the congenital origin, founded his conclusions on the measurements obtained ophthalmoscopically during life of the opaque zone of ten lenses compared with the measurements of fœtal, infantile, and adult lenses. Mr. Treacher Collins gives the direct post-mortem measurements by different observers of the opaque area in ten cases also, the two sets of measurements being as follows :

	Direct measurements (Quoted by Collins). mm.		*Dub's measurements* (Ophthalmoscopic). mm.
Beseline . .	6·	...	4·4
Lawford . .	5·25	...	4·6
Lawford . .	4·5	...	4·7
Lawford . .	4·5	...	4·8
Schreiner . .	5·75	...	4·8
Schreiner . .	3·25	...	5·
Schreiner . .	6·	...	5·2
Collins . .	3·5	...	5·2
Collins . .	3·5	...	5·5
Collins . .	5·5	...	5·6

He also ascertained the average diameter of three healthy lenses at birth to be 5·75 mm., and Dub found the average of three lenses under a year old to be 7·46. Mr. Collins points out that the diameter of the opaque zone is never larger than that of the lens at birth, and concludes, with Dub—putting aside the theory that contraction of the nucleus is sufficient to account for the difference in dimensions—that the lesion is either produced before birth, or else the part affected is not the most peripheral at the time of its occurrence.

I will now endeavour to elucidate this point by evidence derived from an examination of certain peculiarities in the permanent dentition frequently associated with lamellar cataract. This condition has been investigated by many observers, but especially by Grevers (3), Zsigmondy, and

Leon Williams, and has been well named by the first "hypoplasia." The teeth so affected were formerly known as "honeycombed" teeth.

The tooth is marked by transverse lines of depression in which the enamel is seen to be deficient, discoloured, and imperfectly formed; in the less severe cases the enamel may only be marked by small pits or depressions usually arranged in horizontal lines. With the exact character and situation of these lesions I will deal later. That the dentine also is affected is seen when the tooth is examined in section. Seen microscopically, the enamel appears thin in the region of depression, and in parts may be entirely absent. The dark lines found in normal enamel, known as the striæ of Retzius, and supposed to represent the outer surface of the enamel at an earlier period of development of the tooth, are found in abundance and with marked characteristics. These lines, as pointed out by Grevers, follow the "incremental" lines of enamel growth running in a direction downwards and inwards towards the neck of the tooth; they are said to run at an angle of 30° to 35° with the periphery of the dentine. The dentine is also characterised by large numbers of interglobular spaces; indeed, the whole appearance, macroscopic and microscopic, is obviously indicative of imperfect calcification—a condition in which the calcospherites have not completely coalesced to form the normal tissue.

Leon Williams (4) has investigated very completely the histology of these teeth, both in man and animals. He has prepared a large number of specimens showing the condition of the enamel in the neighbourhood of the sulci, where it is incompletely formed, and in the slighter cases, where impaired translucency and pigmentation are the only characteristics visible to the naked eye. In the latter the most obvious features are marked striation of the individual enamel prisms, and stratification in the layers of deposit of the enamel combined with increased pigmentation and granularity. In some cases deflection of the enamel prisms at the lines of stratification is clearly visible. In the former it is seen that the fissures in the

enamel frequently expand on reaching the outer surface of the dentine into flask-shaped spaces—a condition partly due to incipient caries. In other situations in the vicinity granular amorphous masses of enamel exist, from which tracts of imperfect enamel lead towards the surface of the tooth. In thus directing his attention to the investigation of those teeth which to the naked eye appear but slightly affected, Mr. Williams has shown that even in these the structural defect may be really very great, and also that between the least and most severe cases the difference is one of degree and not of kind.

It is important further to consider accurately the periods of incidence of this inhibited growth, as shown by the position of the marks upon the teeth. The dates of calcification of the two dentitions have been investigated with great care by Röse (5). It is with the permanent dentition we are chiefly concerned. He describes the condition at birth as follows:

The first permanent molar in the lower jaw has five well-developed coronal tubercles, but only one calcified cusp. (Röse infers that the calcification of this tooth begins about the time of birth.)

The germs of the incisors and cuspids are in their little sacs (*Zahnsackchenen*), but there is no *Anlage** of the bicuspids.

Of the rudiment of the second permanent molar Röse agrees with Magitôt in finding no trace. He also states that the upper and lower corresponding teeth are developed approximately synchronously.

The condition of the milk dentition at birth is as follows: —Incisors $4\frac{1}{2}$ mm. calcified; cuspids $2\frac{1}{2}$ mm. calcified; first molar 2—3 mm. calcified; second molar two out of five centres of calcification coalesced $1\frac{1}{2}$—$2\frac{1}{2}$ mm. high.

History of the first permanent molar.

Embryo.

17 weeks.—18 cm. long. The papilla projects itself into the thickened posterior end of the *Zahnleiste*† sideways.

* *Anlage* is nearly equivalent to "groundwork" or "rudiment."

† *Zahnleiste* is a word meaning the involution of epiblast which extends

20 weeks.—24 cm. The *Anlage* of the tooth sac appears.

24 weeks.—30 cm. Tooth already well developed and separated from the *Zahnleiste*.

Birth.—One point of calcification present.

4 months.—4 points of calcification 1—2½ mm. high, upper jaw.

6 months.—4 points of calcification 2—3½ mm. high, lower jaw.

10 months.—4 points of calcification fused 5—6 mm. high, upper jaw.

1½ years.—5 mm. high, upper jaw.

2 years.—Root ⅛ mm. long, upper jaw.

3¼ years.—Root 1½ mm. long, upper jaw.

History of the second permanent molar.

4 months.—End of *Zahnleiste* thickened.

6 months.—Papilla inserts itself sideways.

1½ years.—Tooth sac formed.

2 years.—4 points of calcification 1½ to 2 mm. high.

3¼ years.—Crown 3¼ mm. high. Third molar, end of *Zahnleiste* thickened.

5 years.—Papilla presents itself sideways.

The remaining teeth of the permanent dentition, of course, intervene between these extremes, the centrals commencing to calcify at two or three months, and having their crowns completed by about three years. To summarise these observations, then, any cause tending to inhibit calcification during the first three years of life might be expected to show its effects on some part of the crowns of the first permanent molars, and on corresponding parts synchronously calcified of the incisors, cuspids, and bicuspids. Any cause operating later than the second year might affect either these latter or the second permanent molars. The only parts of the temporary teeth likely to show the effect of post-natal affection would be the cuspids and molars in the region of the gum margin.

as a curved flange round each embryonic jaw, and from which the enamel organs of the teeth are derived.

I have examined twenty-six cases of lamellar cataract with the object, among others, of obtaining accurate records of the parts of the teeth affected. Of these, four showed no abnormality of dental tissue. The remaining twenty-two showed lesions of varying extent and position, but with a certain uniformity observable throughout. I do not wish to suggest that this proportion represents that usually obtaining in any large number of these cases, because, probably, those showing dental lesions would be more likely to be sent by those ophthalmic surgeons who were kind enough to refer their cases to me.

Nevertheless the fact that I have been able to collect in some three years, from four or five sources, between twenty and thirty cases of a comparatively uncommon disease in which the dental lesion did exist, together with the ocular lesion, appears to be evidence that such a simultaneous occurrence is not, and cannot be, purely fortuitous, but must point to some causal connection between the two, and in all probability one common cause producing the dual lesion.

I will now summarise the conditions found in these twenty-two cases, concerning which the details appear in the appended table. On one point their evidence is unanimous; the first permanent molar, except in one case in which all four had been removed, and one in which they were completely broken down, showed a deficiency always affecting the coronal surface, and extending for a variable extent down the labial and lingual surfaces. In the majority of cases (twelve) the defect extended either quite up to the gum margin, or to within about an eighth of an inch of it. In the other nine cases the imperfect calcification was neither so marked in extent, nor did it last for so long a time. It is noticeable that the most protracted cases are usually the most severe, but that in all cases, and especially in the most severe ones, the epoch of remission is very clearly indicated by a definite line of demarcation between the bad enamel and the good, there being a marked shoulder where the perfectly formed enamel is first

deposited. This definite ledge of enamel, however, is partly accounted for, no doubt, by the obliquity of the "incremental" lines of the enamel referred to above. That is to say, it would be expected that a groove on the lateral surface of a molar would be undercut on its gum-marginal aspect.

In the nine less severe cases, as a rule, not more than a half to two-thirds of the crown is affected, and in the greater number considerably less, a few cases having the coronal surfaces only abnormally flat and finely pitted. It is distinctly to be observed in the majority of first permanent molars of all cases, however, that the coronal cusps stand up as spikes distinct from the rest of the crown, strongly indicating that the disease attains its maximum intensity not till after these are formed, or at earliest while they are forming. Indeed, in a few cases the cusps may be said to be normal, or very nearly so.

The upper central incisors in all cases show hypoplasia of enamel to an extent which is chronologically in accord with that shown by the first permanent molars. In one case a "terraced" condition with two ridges is observable, indicating an exacerbation of the determining cause at one period. The most important fact, however, is that in only one case was the cutting edge of the tooth anything like normal; in this case there was a transverse depression across the labial surface of some depth at about $\frac{1}{3}$ of the length of the crown from the cutting edge. Above this is a sudden ledge of good enamel, but below the transition to good enamel is gradual, and only just the edge itself can be said to be normal. The full import of this observation will appear when I compare these facts with those derived from an examination of defective teeth not associated with lamellar cataract. Unfortunately in this case all four molars were extensively decayed; it would have been interesting to see whether, as one would have expected, their coronal surfaces were more perfectly calcified than in any other case.

The upper laterals and cuspids are usually affected to an extent corresponding with the central defect; in slight

cases the lateral escapes, but very rarely the cuspid, the cusp of which is apparently calcified very early. In a few cases the tip of the cusp was nearly normal, there being a ring-like depression just above it.

The lower incisors and cuspids are almost always affected in a manner, and to an extent, to be expected from an observation of the upper, but the normal chronology of development appears to be more regular and certain in the lower than in the upper. In three cases only are any marks on the bicuspids to be noticed, and then for the most part in the form of depressed cusps; in one case, however, the upper first bicuspids were hypoplastic to the extent of $\frac{2}{5}$ of their crowns, and lower first bicuspids to the extent of $\frac{1}{3}$. In no case was a second permanent molar affected.

The temporary dentition does not usually come under observation in these children, but slight indications of the condition could be seen in two cases in the deciduous molars. Of course the existence of normal enamel in the anterior teeth and in part of the molars would be interesting as good negative evidence of pre-natal affection.

If all these facts be studied in conjunction with the dates and periods of calcification as investigated by Röse, certain conclusions appear inevitable. The condition of molars, incisors, and cuspids alike suggests the inference that some cause acts to the detriment of their calcification from very shortly after birth until about two years of age usually, or considerably less in many cases. Whatever may be the extent of variability of the date of remission, the transition to normal enamel is nearly always sudden. The onset, on the other hand, shows no such narrowly defined epoch, but it is limited to a few weeks or months in its variability of date. It may be contended that, as most cases show an imperfection of the earliest formed enamel of the first formed permanent teeth, the cause may have its beginning at a period antecedent to this and before birth. I think, however, the occasional cases in which just the cusps of the molars are normal or nearly so,

and the case in which the cutting edges of the central incisors were normal, afford evidence strongly antagonistic to this view.

In any case, if it be admitted, as I think it must, that the frequent co-existence of lamellar cataract and hypoplastic teeth points to a common cause for the two lesions, and if it be proved that it acts for the first two years of life in occasioning the one lesion, it is, to say the least, improbable that it brings about its ocular results by acting before birth, especially when it is remembered that hypoplastic permanent teeth are frequently, if not usually, preceded by a normal deciduous dentition.

I will now discuss the various causes that have been assigned for lamellar cataract, and endeavour to bring fresh evidence for and against those causes. Perhaps more than any other, rickets has been set down as the most probable cause. Mr. Treacher Collins (6) read a paper on this subject before the Ophthalmological Society, in which he dealt with the evidence derived from statistics as to the frequency of rickets and lamellar cataract in various colonies and in Persia. In summing up his paper he said that in Adelaide rickets was a rare disease, and lamellar cataract very infrequent. In Melbourne rickets was until recently comparatively rare; it was more common now, but the severity of the affection was much less than in the old world; lamellar cataract was exceedingly rare, and the "honeycombed" condition of the teeth not often met with. In Sydney rickets was said to be as common as in England, but there was no statement as to its comparative severity; lamellar cataract was less frequent than in this country. In Persia rickets and lamellar cataract were both very rare. The general trend of these conclusions is to show that in the colonies both diseases are rarer or less severe than in this country, but this is, of course, only very slight evidence as to a possible causal connection between them.

In my twenty-six cases I have only been able to obtain a history of rickets in three cases. In one of these there was a definite history of rickets up to three years of age,

but the teeth showed good enamel in that part deposited between about the first and fourth years.

I shall bring evidence directly in favour of the view that rickets rather than being a cause may be a concomitant, and a result of the same general conditions which occasion lamellar cataract.

Further to elucidate this point I have examined a large number of cases of hypoplastic teeth not associated with lamellar cataract, in order to note any differences in character between the two classes. Various causes besides rickets, local and general, have been assigned to this condition,—to wit, exanthematous fevers, convulsions, stomatitis, the exhibition of mercury, and artificial feeding. There is a consensus of opinion among the majority of medical writers that rickets causes delayed and irregular eruption of the deciduous teeth, and that these teeth are rocky, pitted, and deficient as regards their enamel. The major portion of the crowns of these teeth is, however, calcified before birth, the process commencing as early as the third month, so that infantile rickets could not be instrumental in modifying the structure of the enamel. It could only be the necks of the cuspids and molars which might be affected by disease commencing at birth.

Denison Pedley (7), in remarking on the analogy in the case of congenital syphilis, states, from the examination of a large number of children, that while the majority of such had normal teeth, in some cases the necks of the cuspids were affected, either by the disease itself, or by the mercury given in course of treatment. Hypoplastic enamel in the parts of the teeth formed before birth is certainly rare, if existent, in any children, and carious enamel may easily be mistaken for it. George Carpenter and Denison Pedley (8) examined 500 children with well-marked rickets, and found normal teeth in the majority.

I will now discuss the other alleged causes. W. B. Paterson, who examined 339 boys, all of about the age of fourteen, on the training ship "Exmouth," found forty-nine cases of hypoplasia—a proportion of about 14 per cent.

Sidney Spokes (9) gives the results of an examination of a large number of boys, both at a large public school and a large Poor Law school, the percentage being far greater in the latter—12 per cent. as against 4 per cent.

I have myself examined 138 boys at a Poor Law school, of which thirty-one had hypoplastic teeth—a proportion of about 12 per cent. It is evident, then, that among the less well cared-for classes this condition is by no means an uncommon one, and according to Grevers, of Amsterdam, it is even more common in some parts of the Continent.

This fact, taken in conjunction with the fact of the comparative infrequency of lamellar cataract, and the frequent or constant association of hypoplastic teeth with that lesion, is strongly suggestive of the hypothesis, that of several causes capable of producing this type of tooth, one or more only can occasion the ocular lesion.

A comparison of the observations on the teeth of the twenty-two cataract cases, and of the thirty-one cases from a Poor Law school, affords remarkable differences. Whereas the former practically all show an affection, commencing, presumably, shortly after birth, the latter only afford sixteen which can be placed in this class. Even of these four are so very slightly marked as hardly to justify their inclusion, less marked by far than any of the cataract cases.

As regards duration, these sixteen cases also closely resemble the cataract cases. It is interesting to note in one case the crowns of the second molars were distinctly pitted —a condition I have observed in two other instances, and of which Berten has recorded three examples. On the other hand, there are eleven cases in which at least the cusps of the molars, and, in the greater number of cases, a large part of the crown of the molars or part of the incisors are normal, there being a horizontal line at a part of the tooth corresponding to an age certainly several months or even two or three years after birth. Three other cases show marks only on single teeth, and are not included on account of the probability of local affection

being the cause of the condition. The important point to observe is that this type of tooth, which is frequently seen, is, so far as I know, not associated with lamellar cataract, and it is therefore highly improbable that all hypoplastic teeth are produced by a common cause.

In seeking for these causes in the histories of individuals vagueness takes the place of definite observation, but in many cases the history is sufficiently clear and precise to be credited.

I have endeavoured to obtain such history in all the cataract cases, and I have also notes on a number of dental cases mostly from my own observations.

It has been thought that exanthematous fevers are responsible for some of these cases, and Smale and Colyer (10) quote a case in which a child had an attack of scarlet fever at two years of age, followed very shortly by measles. Upon the lingual surface of the centrals and laterals there were found after eruption two transverse marks rather more than halfway up the tooth. C. A. Harris (11) also records a very interesting case of a girl nine or ten years of age. The upper centrals showed a pitted incisive edge, two or three dark marks a little further towards the gum margin, and a distinct groove about an eighth of an inch from it. The laterals showed a groove rather more than an eighth of an inch from the gum margin. The lowers were marked in corresponding positions, and the first permanent molars had a line about halfway between the coronal surface and gum margin. The child had suffered from measles at twelve months, scarlet fever at sixteen months, and dysentery at twenty-two months.

Many of my cases support this view. I cite the following as affording definite histories of some acute illness associated with defective teeth. The measurements are not exact, as such would be misleading owing to the variable size of similar teeth and their frequently stunted condition, but are given as estimated ratios in relation to a complete tooth.

(1) Centrals transverse line $\frac{1}{3}$ from cutting edge; other teeth to correspond. Lacrymal abscess two years of age.

(2) Slight double line on centrals close to gum; laterals, cuspids, and first bicuspids to correspond. Nearly died of measles between four and five.

(3) Double line on first bicuspids, upper $\frac{3}{4}$ from incisive edge. Measles between six and seven.

(4) Upper centrals, broad line $\frac{2}{5}$ from incisive edge; laterals normal; cuspids, spiky cusps; lowers similar. Measles eight months old.

(5) Second line on previously affected centrals close to gum; also on lower centrals and laterals. Slight whooping-cough at five.

(6) Fine dotted line centrals, $\frac{2}{3}$; laterals, $\frac{1}{2}$; cuspids, $\frac{2}{3}$; first bicuspids and second molars also affected. Measles between three and four.

(7) Centrals, line across centre; laterals deficient, cutting edge and line $\frac{1}{3}$ from edge; cuspids deeply pitted to $\frac{1}{3}$; lowers similar; six year molars normal. Scarlet fever between three and four.

(8) Centrals, line across centre. Lateral has lower portion irregularly pitted and discoloured. Apices of cuspids similar. Patient always delicate, measles when young, at what age uncertain.

(9) Centrals reduced to stumps. Laterals and cuspids pitted *towards neck*, tips gone. Lower incisors ridged for $\frac{1}{3}$ *from gum margin.* Cuspids $\frac{1}{3}$ from cusp. Healthy until measles at three, followed by scarlet fever. Fits ever since.

It is curious to note that there is no case of a molar being marked by a transverse line on its lateral surface. A large number of cases might afford such, but the general observation in conjunction with the case in which, although the centrals were affected by a mark across the centre, the molars are noted as normal, suggests that these teeth, although easily affected in early infancy, are more resistent than other teeth to noxious influences of later date.

The positions of the marks in all these cases are not absolutely consistent, but it must be remembered that there are great differences in the periods of eruption of permanent teeth and presumably in their development. The relative

periods of eruption of the teeth of a single dentition are also much influenced by the loss or retention of the deciduous predecessors.

All these cases are of teeth affected at comparatively late periods. It will be profitable to compare with them some cases of teeth affected shortly after birth.

(1) Molars, $\frac{1}{2}$; centrals, $\frac{1}{4}$; cuspids, tip; laterals normal; lowers similar. Always bad health. Trouble cutting teeth, treated for it. Croup at one year (*circa*). Scarlet fever under two.

(2) Molars, flat crowns, spiky cusps; normal line, $\frac{1}{8}$ in. from gum; centrals, $\frac{1}{3}$; lowers similar. At eight months bronchitis, inflammation of lungs and whooping-cough; twelve months measles.

(3) Molars, $\frac{1}{2}$; centrals, $\frac{1}{3}$; others similar. Always rickety and small. Medicine from nine months.

(4) Molars, $\frac{1}{2}$; centrals, $\frac{1}{3}$; laterals and cuspids, cusps. Artificial food from infancy. Fits when teething and very ill eight to twelve months.

(5) Molars, $\frac{1}{3}$; centrals, $\frac{1}{4}$; cuspids, cusp; lower incisor and cuspid, $\frac{1}{5}$. Fit when teething. Whooping-cough, two and a half; measles three.

(6) Centrals, $\frac{4}{5}$; laterals, $\frac{1}{2}$; cuspids, $\frac{3}{5}$; first bicuspid, cusp; lowers similar. Teething powders. Measles eighteen months.

(7) Centrals, $\frac{4}{5}$; laterals, $\frac{4}{5}$; cuspids, $\frac{1}{2}$; lower incisor, $\frac{3}{4}$; cuspids, $\frac{1}{2}$. Fits at two. Powder from doctor. Measles three and a half. Scarlet fever five.

(8) Centrals, line near edge; laterals, line near edge; cuspids, cusp affected; lowers similar. Teething powder. Measles and thrush when an infant.

(9) Molars, coronal surface; centrals, a few pits. Artificial feeding. Convulsions first at four months.

(10) Molars, $\frac{2}{3}$; centrals, $\frac{1}{2}$; others similar. Teething powders.

These are fair examples of the kind of history one may frequently obtain in those cases affecting the teeth early. It may be stated generally that while it is not unusual to

get a definite history clearly pointing to the cause of the lesion in those less frequent cases where the teeth are affected after part has been correctly formed, on the other hand in those more frequent cases in which the affection commences immediately after birth, definite illnesses are not commonly traceable, although conditions of general disturbance of health for which evidence is afforded by convulsions may often be found.

Two final cases are interesting as clear examples of a succession of affections producing a series of marks.

(1) Molars, flat with spiky cusps, having nearly normal enamel; buccal surfaces, "terraced;" upper centrals, pitted edge; line across centre and fine line close to gum margin. Teething powders at eight or nine months; breast-fed until two; convulsions for one year during teething; measles one and a half; scarlet fever three years.

(2) Lower molars, flat crowns pitted and pigmented with small cusps of enamel; ridge at about halfway between coronal surface and gum; slight pitted line, $\frac{1}{16}$ in. further towards gum; uppers, similar crowns and very faint line $\frac{1}{16}$ in. from gum margin. Upper centrals, deficient enamel, irregular edge and surface for $\frac{1}{3}$, then ridge of better formed but imperfect enamel, then another depressed line at $\frac{2}{3}$; laterals, short with irregular edge and line at $\frac{1}{2}$; cuspids, unerupted; lower centrals and laterals, similar to upper; cuspids, just erupting with ringed cusps. Born with "consumptive bowels." Fits during early infancy and again at a year and nine months. Always been very delicate. Whooping-cough and measles at about three years. Temporary molars marked with slight ridge on lingual surfaces close to gum margin, the thick edge of enamel usually found in temporary teeth being present, however, beyond.

The existence of imperfectly calcified teeth in animals requires, as Leon Williams has pointed out, other explanations as to its cause than that afforded by exanthematous fevers, and is, I think, indicative of a more generalised cause such as I suggest.

Taking now the twenty-two cases of lamellar cataract, the teeth of which, as I have said, resemble the latter class, I find that as many as thirteen had convulsions in infancy. Three had a distinct history of rickets, but not always at an age early enough to account for the cataract. In one there was an account of general bad health from infancy. Two gave definite histories of artificial rearing, and doubtless many of the others were similarly reared. A few had received teething powders, two had had measles, and in three cases absolutely nothing bearing on the point was elicited.

It should be stated that in a large number of cases of very defective permanent teeth, probably including many in which cataract co-exists, it is impossible to obtain any definite history, and, on the other hand, there must be innumerable instances of infantile disease in one form or another in which no evil results to enamel or lens follow. In connection with this latter point an analogy may be reasonably drawn with congenital syphilis. Although the notched, peg-shaped, permanent incisors are regarded as diagnostic, or at least markedly characteristic, of the disease, their frequency, relatively to the number of cases in which they might conceivably occur, is almost infinitesimal.

The general trend of these observations, then, goes to show that hypoplastic teeth may be produced by a variety of causes of which exanthematous fevers certainly form one class. Convulsions are certainly a frequent concomitant in the class of cases occurring early, and this fact would appear to indicate that no single illness is to be looked for, but a condition of general ill-health and digestive derangement brought on, no doubt, in many cases by errors in feeding. The fact that this condition is more frequent in those classes of society where children receive least attention and most injudicious treatment supports this view. It is in this type of children that rickets would be likely to declare itself later, and I believe that the lamellar cataract, with which this class of tooth is frequently associated, and

the rickets are produced by the same general cause as the dental lesion.

The view which has been put forward that stomatitis may be a cause appears to me completely untenable; were it a cause a large percentage of children would be affected for months together with stomatitis, whereas ulcerative stomatitis of any degree of severity is by no means a common affection. Tomes (12) quotes a case in which the patient when an infant was heavily dosed with calomel; the teeth, however, may have been affected by the disease for which the mercury was given. Although the exhibition of mercury may be an occasional cause, this drug is not now sufficiently used to account for many cases, and, if in the form of teething powders, is given too late to produce the marks in the position in which they are seen.

J. Kingston Barton (13) states that in 202 children he found 10 cases of defective permanent teeth and 5 temporary. The latter occurred out of 67 hand-fed children, 24 breast-fed showing nothing. An infant temporarily hand-fed at six weeks showed a horizontal line of defective enamel.

Observations on the deciduous dentition afford inconclusive inferences. Temporary teeth are generally said to show defective enamel about half as frequently as permanent; but, as far as my observation goes, it is very rare to find the temporary teeth marked in the clearly defined synchronous manner in which the permanent ones are, although the enamel scarcely appears normal and may be irregularly pitted. Observation of temporary teeth is rendered very difficult by co-existing caries. Further, they are almost completely formed by four months after birth, so that one would only expect the deciduous cuspids and molars to show much sign of post-natal disorder. Definite examples of hypoplastic temporary incisors would be of much interest. I have noted in one case a distinct groove about $\frac{1}{8}$ in. from the incisive edge of the lateral incisors, the centrals having been shed.

To sum up, then, these conclusions, I think it is clear

that lamellar cataract is a disease of early infantile life, that the probable cause is some general derangement of health which also affects the teeth, the most probable being errors of feeding and nutrition. It is worth remembering that the dental enamel and the lens are each formed from an involution of epiblast, which eventually becomes isolated within mesoblastic tissue, and that it is therefore not improbable that both should be affected by the same general condition of ill-health.

These conclusions would appear, at first sight, not to be consistent with those afforded by the measurements of lenses by Bernard Dub, Collins, and others, who showed that the diameter of the opaque zone was never larger than that of the lens at birth. From this fact Bernard Dub drew the conclusion that the zone of opacity was either formed before birth, or was not the most peripheral at the time it became opaque.

Mr. Treacher Collins, however, in reply to the discussion on the paper referred to above, read before the Ophthalmological Society, said that he thought this assumption was not necessary, but that it was quite likely, as the result of some general disturbance of nutrition, that the part of the lens furthest from the nutrient supply should contract, and, as the result of such contraction, that an opaque zone should be formed between the nucleus and the cortex from which it had contracted. Microscopical examination of lenses affected by lamellar cataract had shown that the morbid changes in them were not confined to the opaque zone, but that the nucleus was also involved.

Should my conclusions be confirmed by further observations on the hypoplastic teeth associated with lamellar cataract, the deduction would appear inevitable, if these lens measurements are to be relied on, that the ocular lesion differs from the dental lesion in being produced after the development of the tissue instead of during that process, as it must be in the case of a calcified tissue.

In concluding this paper, I should like to thank those gentlemen who have kindly sent me cases for examination,

and especially Mr. Grimsdale and Mr. John Griffith, to whom I am greatly indebted. I need hardly say that if any members present should feel inclined to make further observations on the teeth of those cases of lamellar cataract which come under their care, I should always be pleased to render any assistance in my power.

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(*November 8th,* 1900.)

Measurements of Hypoplastic Portion of Teeth associated with Lamellar Cataract.

Class I.—*First permanent molars affected to within about $\frac{1}{8}$ in. from gum margin.*

No.	Upper. Centrals.	Upper. Laterals.	Upper. Cuspids.	Lower. Centrals	Lower. Laterals.	Lower. Cuspids.	Remarks on 1st perm. molars.	Other teeth.
1	$\frac{3}{5}$	$\frac{1}{2}$	Not erupted.	$\frac{3}{5}$	$\frac{4}{5}$	Not erupted.	Spiky cusps	2nd temp. molars pits and flat cusps.
2	$\frac{4}{5}$	$\frac{3}{5}$	Not erupted.	$\frac{4}{5}$	$\frac{4}{5}$	$\frac{4}{5}$	Spiky cusps	—
3	$\frac{5}{6}$	$\frac{1}{2}$	$\frac{5}{6}$	$\frac{2}{3}$	$\frac{2}{3}$	$\frac{1}{2}$	Cusps nearly normal . .	—
4	$\frac{2}{3}$	$\frac{2}{3}$	$\frac{5}{6}$	$\frac{5}{6}$	$\frac{5}{6}$	$\frac{1}{2}$	Horizontal ridges to gum margin	1st upper bicuspids notched outer cusp.
5	$\frac{2}{3}$	Short and notched.	$\frac{1}{3}$	$\frac{2}{3}$	$\frac{1}{2}$	$\frac{1}{3}$	Spiky	—
6	$\frac{5}{6}$	$\frac{1}{2}$, short thick edge	$\frac{1}{3}$	$\frac{1}{5}$	$\frac{1}{5}$	$\frac{1}{2}$	Some good enamel on cusps .	—
7	$\frac{2}{3}$	$\frac{1}{2}$	$\frac{1}{3}$	—	—	$\frac{1}{3}$	—	Four bicuspids depressed, cusps, one grooved near neck.
8	$\frac{2}{3}$	$\frac{1}{2}$	$\frac{1}{3}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{3}$	Nearly to gum margin . .	Upper 1st bicuspids $\frac{2}{5}$, lower 1st bicuspids $\frac{1}{3}$.
9	Half erupted, deficient enamel	Not erupted	—	—	—	—	Sharp cusps	—
10	$\frac{1}{2}$	Normal enamel, but short	$\frac{1}{3}$	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{3}$	Cusps normal . . .	—
11	Very short, with pitted edge	Normal	$\frac{1}{6}$	Very short with pitted edges.			—	—
12	$\frac{2}{3}$, two ridges	$\frac{1}{3}$, one ridge.	$\frac{1}{2}$, second earlier, ridge faint	$\frac{2}{3}$ each with earlier ridge	$\frac{2}{3}$	$\frac{1}{2}$	Extracted.	—

CLASS II.—*First permanent molars affected to the extent of two-thirds of crown or less.*

No.	Upper.			Lower.			Remarks on 1st perm. molars.	Other teeth.
	Centrals.	Laterals.	Cuspids.	Centrals.	Laterals.	Cuspids.		
1	Rather short, dotted line near edge	Normal	Similar to central	Each has dotted line near edge			$\frac{1}{2}$, a cusp nearly normal . .	—
2	None of these teeth erupted.			None of these teeth erupted			$\frac{2}{3}$ defective spiky cusps .	Temp. molars show signs of deficient enamel.
3	Pitted edge	Rough edge	—	Normal	Normal	—	Uppers have line of small pits, lingual surface. All have slightly defective crowns	—
4	$\frac{1}{8}$	Normal	Normal	$\frac{1}{8}$	$\frac{1}{10}$	Ringed	$\frac{2}{5}$, finely pitted . . .	—
5	Short, edge pitted	Normal	Depressed cusp	Thick, pitted edge, line across centre	Thick, pitted edge	De-pressed cusps	—	—
6	$\frac{1}{5}$	Normal	Ringed cusp	$\frac{1}{5}$	$\frac{1}{5}$	$\frac{1}{6}$	Short, pitted crown . .	—
7	Line $\frac{1}{3}$, good ine. edge	Line $\frac{1}{4}$	Line, $\frac{1}{5}$ good cusp	Line only			Decayed, broken down . .	—
8	$\frac{1}{3}$	—	Normal	$\frac{2}{5}$ $\frac{1}{6}$	$\frac{2}{5}$ $\frac{1}{6}$	$\frac{1}{3}$ Cusps pitted	Ridge at $\frac{1}{2}$	—
9	$\frac{1}{3}$	$\frac{1}{4}$	$\frac{1}{5}$	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{4}$	Ridge $\frac{1}{2}$ to $\frac{2}{3}$ from coronal surface, spiky	—
10	$\frac{1}{4}$, second line $\frac{2}{3}$	Normal.	$\frac{1}{5}$	$\frac{1}{4}$, second line $\frac{2}{3}$	$\frac{1}{4}$	$\frac{1}{5}$	Broken down . . .	—

The President asked whether Mr. Bennett had, in these cases, inquired into the family history. A few months ago he showed at the Society a boy with lamellar cataract, for which he performed an optical iridectomy. The father suffered from a lamellar cataract, as also did two children, seven cousins, one niece, and two sisters, all belonging to one family. In view of that series it would be useful if attention could be directed to this feature and a note made of it.

Mr. Grimsdale said he believed that one case which he sent to Mr. Bennett had a history of that kind. He thought they could trace eight cases in three generations.

Mr. Treacher Collins said he was very much interested in Mr. Bennett's valuable paper, with the conclusions of which he was in complete agreement. With regard to the vexed question of the congenital or infantile origin of lamellar cataract, it occurred to him (Mr. Collins) some time ago that it would be interesting to know what was the condition of the permanent teeth in cases of cataract which were definitely of congenital origin. He could find no reference in the text-books concerning it; therefore he collected a number of cases which had been operated on for congenital cataract, and who had since reached the age at which the permanent teeth would be developed. He had now examined ten such cases, but in none of them was there any defect in the enamel of the teeth such as was found in connection with lamellar cataract.

Mr. John Griffith said he would like to ask Mr. Bennett when the cells were laid down for the formation of the enamel. He (Mr. Griffith) believed that they were developed about the sixteenth week of fœtal life, therefore he did not see why there should not be an error in the growth of those cells—the adamantoblasts—which formed the enamel, instead of an error in calcification. If the adamantoblasts were imperfectly developed, there would be a corresponding defect in the enamel of the permanent teeth. Some time ago he showed before the Society cases of absence of the iris in two children. In one there

was a history of fits, and hypoplasia of the dental enamel. He was inclined to look upon lamellar cataract as a congenital disease, not as infantile.

Mr. Bennett, in reply, said, referring to the remarks of the President and Mr. Grimsdale, it would be interesting and important if observers would note whether more than one case occurred in the same family, and whether the teeth were also involved, because there might be the one cause operating throughout. Mr. Treacher Collins's cases of true congenital cataract were extremely interesting, because they afforded a kind of negative evidence in proof of the infantile origin of lamellar cataract. It meant that in those cases of obviously congenital origin, from the ocular standpoint the teeth were absolutely normal. With regard to Mr. Griffith's remarks, he did not think the conclusion drawn by that gentleman was altogether a reasonable one. He understood Mr. Griffith to mean that the cells which produced the enamel might be in some way injured before birth, so that they would not produce normal enamel afterwards; if that were the case they would never produce normal enamel at any time. One nearly always found that a certain portion of the enamel formed at a certain date was perfectly normal; this could not be so if the ameloblasts were injured.

VI. DISEASES OF THE RETINA AND CHOROID.

1. *Superficial choroidal atrophy, without subjective symptoms, in a member of a family subject to night blindness.*

By A. Hugh Thompson, M.D.

Mrs. D—, æt. 57 years. In each fundus there is an area of superficial choroidal atrophy round the disc, with a well-defined margin, the whole area, including disc, being about two disc diameters, more or less. Beyond this area is a good deal of pigment disturbance, the macula being comparatively normal. The changes are remarkably symmetrical in the two eyes.

V. R. $\frac{6}{9}$ (3 letters), not improved by correction + 1 D. cyl. axis vertical.

V. L. $\frac{6}{6}$ (3 letters), H. 0·5.

Fields very nearly full. No scotoma. No night-blindness. Fields quite as full in a dim light with a grey test-card as in a bright light with a white test-card.

Patient says that *her father*, who died thirty years ago, always stumbled against things, on account of bad sight, and yet could read a small-print copy of the Bible.

Three of his grandsons, brothers to each other, and nephews to Mrs. D—, are affected in varying degrees with night-blindness. *One* of these, who was at Moorfields three years ago, was found to have minute visual fields, with V. $\frac{6}{9}$ in each eye. The *second* was at Moorfields two months ago, where I took notes of his case. He had symmetrical changes in each eye, *viz.*:

(1) Pigment changes at macula.

(2) An irregular but well-defined area of normal choroid, with centre at macula.

(3) Atrophy of chorio-capillaris, with pigmentary changes beyond this area.

Fields full peripherally, but with *ring scotoma*. He complained of night-blindness. His vision was R. — 2·5 $\frac{6}{6}$. L. — 3·0 $\frac{6}{12}$.

A *third* brother is said to be getting to see as badly as his grandfather, and to be afraid to go out after dark.

There is no history of consanguinity, and the health of the family generally is said to be good.

(*Card specimen. December 13th,* 1900.)

The President said he would like to mention a practical point which he had found useful, namely, the employment of a golden-yellow glass in cases of nyctalopia. It chiefly helped in twilight. He was told that it greatly assisted hunters in India, and enabled them to continue their sport for a considerably longer time than they would otherwise be able to. He believed the exact way in which it acted was in neutralising the violet rays.

Dr. Edridge-Green said it would be interesting to know whether the patient saw as well and quickly with the yellow spot as normal-sighted people. Helmholtz had mentioned, stating that the fact was quite unexplainable, that a perceptible interval elapses before we are able to see with the yellow spot. He (Dr. Edridge-Green) thought this was due to the time occupied by the diffusion of the visual purple into the yellow spot. It was easy to see the phenomenon after darkness, as on waking in the morning; on looking at a dimly-lighted surface, the portion of the field of vision corresponding to the yellow spot became marked out as a black patch, and light seemed to invade this patch from without inwards, the centre of the fovea centralis being the last point to convey a sensation of light.

Mr. Nettleship observed that in spite of the widely-spread pigment changes in the patient's eyes the retinal arteries were of good size, and he thought that the good condition of his sight might be largely due to that fact.

It would be interesting to know whether in the grandfather and the nephews vision became worse or remained stationary, as in the patient, and what was the condition of the retinal vessels in them.

Dr. Thompson replied that the grandfather did not go blind. The nephews were getting worse. The retinal vessels of one of the nephews whom he had seen were of fairly good size.

Dr. Tempest Anderson asked whether the wearing of yellow glasses was an expedient arrived at by accident or by design, or was it founded upon the use of the yellow screen in photography? An ordinary photographic plate, if directed to the blue sky and white clouds, would render both as of the same colour. But the employment of a yellow screen and isochromatic plates resulted in the bringing out of the clouds distinctly. That was also useful in certain landscapes, especially where there was a blue haze.

The President, in response to Dr. Anderson's remark, said the glasses were given to him by a gentleman of scientific attainments, who had made the experiment for himself. He suffered from nyctalopia. He (the President) had tried it in other similar cases with considerable success. It also seemed to be useful in some forms of cataract during the twilight hour.

2. *An unusual form of choroiditis.*

By Charles Blair.

(With Plate II, fig. 1.)

Mrs. W—, æt. 42 years, came to the Western Ophthalmic Hospital on November 5th, 1900, complaining of defective vision of right eye. She said she had first noticed it about

a month before, when a veil seemed to gradually come over the sight of that eye. Previous to this her eyes had given her no trouble, and the sight of both had always been good; she had always been healthy but pale. The patient says she has "fibrous tumour of the womb," and suffers from profuse menorrhagia. She was married twenty years ago, and had one child about a year after, which died at two and a half months. There have been no miscarriages, and there is no definite history of syphilis or tubercle.

R. V. $\frac{4}{60}$, not improved, J. 20; L. V. $\frac{6}{6}$, no Hm., J. 1.

In the lower part of the fundus, extending up to the disc, are numerous, closely packed, white patches, some discrete, but mostly confluent, not raised, with sharply defined more or less irregular edges. The patches are especially crowded along the retinal vessels, the vessels passing, however, uninterruptedly over them. The condition is strictly confined to the lower segment of the fundus, the rest being quite normal. There is an almost entire absence of pigmentation, and the vitreous is somewhat clouded with fine dust-like opacities. The field of vision is deficient above.

Urine: sp. gr. 1016, acid, no albumen or sugar.

(*Card specimen. December 13th*, 1901.)

Mr. Jessop would also be glad to hear from any member whether the dust-like opacities and clouds, which were seen to be so numerous in the present case, were found in any condition except syphilis; to him the condition was pathognomonic of syphilis.

Dr. Batten said the condition of the patches in Dr. Blair's case resembled those of a case shown by Mr. Bickerton last session, also one shown by himself two years ago, in which the patches were very much of the same shape, and characterised by the absence of pigment; but in his own case they were symmetrical and far more diffused, and did not affect the vision. In his own case, also, the field of vision was not altered, and probably his condition was congenital. He believed that, in Mr. Bickerton's case, the vision was not affected. One other case was

shown last session of central choroiditis, in which the absence of pigment occurred in the same way. There, again, he believed the vision was not definitely affected. In his own case there were no vitreous opacities, and he thought there were none in Mr. Bickerton's case. The girl had a full vision—$\frac{6}{9}$, and the vitreous was clear.

Dr. C. O. Hawthorne said he had seen a patient within recent months in whom the appearances were practically identical with those in the present case. The patient was about twenty-four years, and the changes were in one eye only; her complaint was, that within the last few months she could not see objects above a certain height. The choroidal changes were limited to the lower part of the fundus.

Mr. Blair, in reply, said that the patient was quite sure that the sight of her right eye remained good up to two months ago; there was then a definite history of commencing defect of sight.

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3. *Injury to an eye by direct sunlight; "eclipse-blinding;" with obstruction of a retinal artery and hæmorrhage into the vitreous.*

By Rayner D. Batten.

(With Plate II, fig. 2.)

The case I report belongs to the class of cases caused by exposure of the eye to the direct rays of the sun, or "eclipse-blinding." The exposure of the retina to the action of direct sunlight appears to produce various degrees of damage; but upon what the degree of damage depends is very uncertain, a quite short exposure in some cases producing extensive damage, whereas in other cases a pro-

PLATE II.

Fig. 1 illustrates Mr. Charles Blair's Unusual Form of Choroiditis (p. 68).

Fig. 2 illustrates Mr. Rayner D. Batten's case of Injury to an Eye by Direct Sunlight (p. 70).

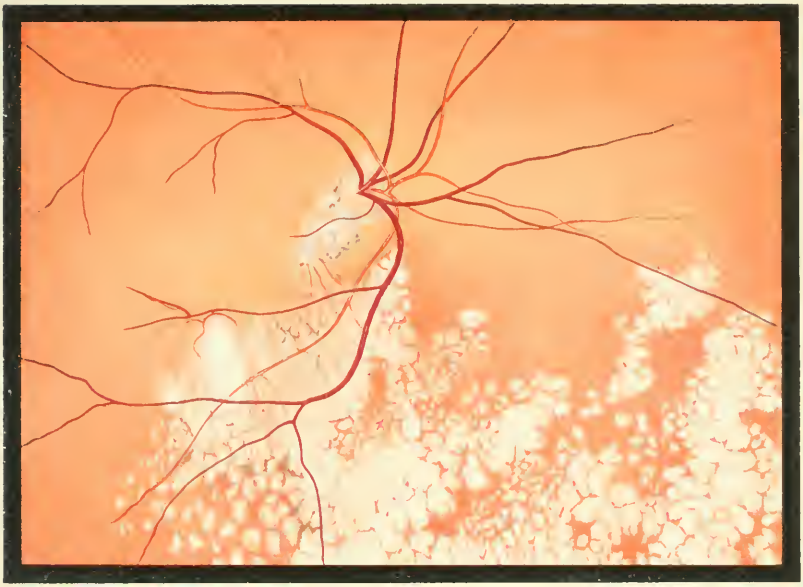
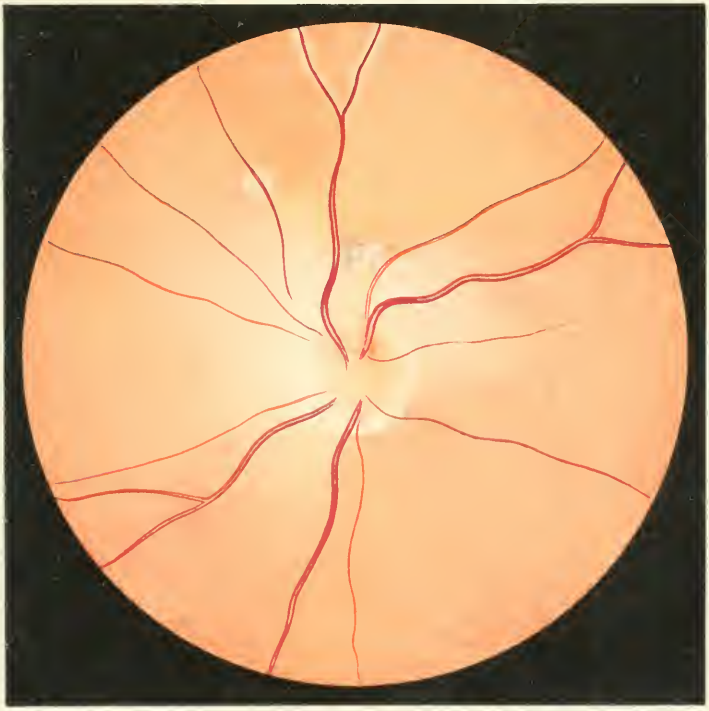


Fig. 1.



M. H. Brooks, *del.* Fig. 2. *Bale & Danielsson, Ltd., Chromo-Lith.*

longed exposure causes no other effect than a temporary central scotoma.

The commonest form of eclipse-blindness is a central scotoma, more or less permanent, with or without definite changes at the macula. But beyond these, there are a few cases reported in which gross injury has occurred, causing permanent loss of vision, and to this class my case belongs.

A woman, æt. 28 years, was sent to me by Dr. Turnbull on June 6th, 1900. She had watched the eclipse on May 28th. She observed it twice, once at about its full, and the second time towards the end of the eclipse. She did not use any protection, except looking between her fingers and screwing up her eyes. She was not conscious of any discomfort at the time, but, on looking down afterwards, "things looked black." The next morning she was conscious of a "black star" in front of her eyes, and could see only portions of things.

On June 6th, when I first saw her, the "black star" had cleared up, but things still looked "misty." She had not clearly distinguished between the two eyes, so that it was not possible to say whether the "black star" was in both eyes or only one. She had some aching in the right, *i. e.* the unaffected eye.

On examination, the right eye appeared normal, the pupil active, the fundus and media normal, except that the veins seemed somewhat enlarged. V. = $\frac{6}{6}$ nearly.

In the left eye, the pupil reacted to light, but was less active than the right, and varied in size. It only contracted fully consensually. V. = $\frac{6}{18}$ with difficulty. There was a total loss of the lower portion of the visual field towards the nasal side (*see chart*). Under homatropine, the pupil dilated evenly. On ophthalmoscopic examination, the vitreous was hazy, with opacities chiefly towards the anterior and lower portion of the vitreous, with one rather large dark-coloured opacity, suggesting a hæmorrhage into the vitreous. The O. D. was very blurred. The central portion was obscured by a whitish haze. The margins of the O. D. were hazy but distinguishable, except at the upper part.

Above the O. D. was a large whitish area, about half the size of the O. D., with ill-defined margins. The patch suggested a retinitis or absorbing hæmorrhage. There were two bright white patches of retinitis to the temporal side of the O. D., and two small fan-shaped patches to the nasal side, probably small hæmorrhages.

The arteries and veins were difficult to distinguish from each other by their colour, both appearing dark. The veins were markedly constricted in places, and enlarged at others. There was a marked constriction of the upper nasal vein just above the O. D. No changes at the macula were detected.

On June 9th, the media were clearer, but the retinitis was more marked, and the O. D. appeared more white and hazy. The retinitis was extending, or becoming more visible, along the upper nasal branch.

On June 12th, the haze of the retina and O. D. had considerably increased, hiding everywhere the margins of the O. D. and some of the vessels, and obliterating the smaller patches of retinitis. A circular, ill-defined, white patch, with a retinal vessel crossing it, was observed for the first time to the nasal side of the fundus.

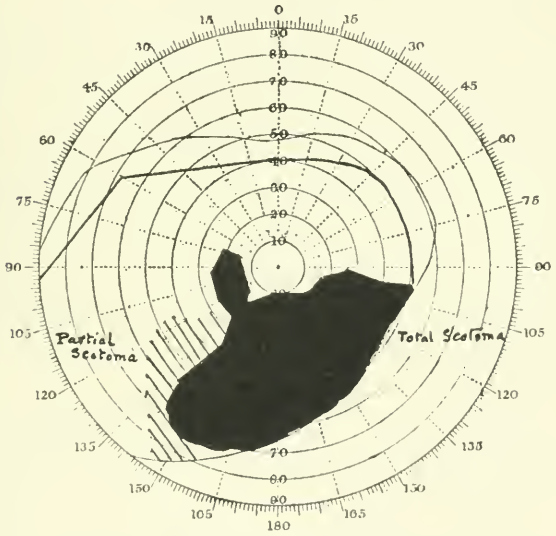
On June 18th, the haze at the upper part of the O. D. had become less marked, but increased at the lower part of the O. D. where the vessels were nearly hidden in the haze. The lower vein was large and distended.

June 28th.—From this date the haze of the retina subsided; the margins of the O. D. became clearer; the vitreous opacities cleared up, leaving only some fine dust-like opacities. As the retinal haze subsided, the ghost of the nasal branch of the upper retinal artery could be distinguished, and traced outwards as a fine line to where it crossed the circular patch of choroiditis already mentioned.

I saw her again on September 27th. She then complained that there was always a "fog" in front of the left eye, which was partly cleared on wearing her glasses, and that lamps at night looked as in a fog.

I last saw the patient on January 9th, 1901. The left pupil was still slightly less active than the right. On examination of the fundus, the œdema had entirely cleared up; there was an irregular patch of choroidal atrophy immediately above the O. D. in the site of the primary lesion. The small patch of choroiditis to the nasal side of the O. D. was more defined, but not pigmented. There were a few small white dots in the neighbourhood. The vessel crossing over it could be traced as a thin line. All the arteries appeared

FIG. 1.



Field of vision of the left eye, June 6th.

somewhat small and reduced in size. No central scotoma was detected for white or for colours. The vitreous opacities had cleared up.

She was treated with blisters to the temple, dark glasses, and later a shade over the left eye. I also prescribed atropine drops, as the hyalitis suggested some damage to the ciliary body.

After leaving off the shade she was much troubled with diplopia; no ocular paralysis could be detected; she proved to be hypermetropic, and I ordered her glasses—right and left + 1·50—which relieved her diplopia.

The vision in the left eye improved from $\frac{6}{18}$ to $\frac{6}{12}$, but appeared to remain stationary at that. The field was taken on more than one occasion, but did not vary materially. There was total loss of the lower nasal field, and partial loss of a small area adjoining it (*see chart*). The area corresponds mainly to the portion of the fundus supplied by the occluded vessel.

I could find no evidence of any central scotoma in either eye, nor any distortion of images, nor any ophthalmoscopic evidence of damage to the macula. This appears to me somewhat extraordinary, especially considering the extensive lesion at the O. D. I would suggest as a possible explanation of the extensive damage to the left eye that the patient, in screwing up her eyes and looking through her fingers, had some excess of convergence, looked at the sun with her right eye, squinted with the left, and consequently brought the O. D. of that eye into the direct line of the sun's rays, and that area being comparatively insensitive to light, had the full force of the sun focussed on it.

As regards the nature of the lesion, the chief (if not the only) one was the thrombosis of the upper branch of the retinal artery, which apparently occurred at, or at all events a few hours after, the exposure. How much of the peculiar white haze was due to the direct action of the sun, and how much to œdema and infiltration of the retina, consequent on the interference with the circulation, I have no means of judging, as I did not see the patient until a week after the occurrence, but from the extension of the haze from one portion of the retina to another, I judge that it was probably due in the main to œdema. There were also various hæmorrhages, probably a large one above the O. D., which was already undergoing degeneration when I first saw the patient, and a few other

small ones. I see no evidence to determine the source of the hæmorrhage into the vitreous, but I think that the vitreous opacities must have been due to a hæmorrhage, as it is difficult to understand how the passage of the sun's rays through the vitreous should have, in themselves, caused the damage.

It is somewhat puzzling to understand why the sun's action should have been so marked in this case. The eclipse, as seen in London, was at no time very bright, the day being somewhat cloudy. As regards the patient, she was healthy, heart-sounds and urine normal. The only history pointing to any peculiar vulnerability of the vessels was that on two occasions, after the extraction of teeth, bleeding was stopped with difficulty.

The use of coloured glasses does not appear to be an efficient protection, although they may cut off enough light to enable the sun to be observed in comfort.

The rapid tolerance of the retina to the sun's rays is a source of danger. After the first effect of glare experienced on looking at a bright light has been overcome, the eye can continue to observe the light without discomfort, and distinguish more detail, and it is probably in this condition of anæsthesia that the damage is done. I think that this may in part be the reason of the greater frequency of sun-blinding from watching eclipses than from other causes. The eye becomes tolerant to the sun's rays during the darker portion of the eclipse, and is consequently anæsthetic, and therefore does not avoid the brighter rays towards the end of the eclipse.

As regards the literature of the subject, I have found little of interest since the able paper published by Mr. Mackay in the *Ophthalmic Review*, Jan., 1894, in which he gives an historical review of the subject, and a summary of all reported cases up to date, with an account of experimental work done on the subject, and some cases of his own.

In the *Ophthalmic Review*, 1895, J. W. Barrett reports a case of a girl, æt. 17 years, who watched the eclipse for a

quarter of an hour through two dark blue glasses, one yellow and one red glass. She acquired a small central scotoma in the left eye, with but little ophthalmoscopic change, the macula appearing only a little larger and darker.

In the *Medical Press and Circular*, 1883, Dr. Drummond reports a case of a girl, æt. 11 years, who, after a "sun-gazing competition" in which she was victorious, having gazed at the sun for twenty minutes, could only grope her way home. Two days later she partially regained her sight. A month after she had headache and vomiting for some weeks, with loss of memory and unconsciousness. Headache and vomiting continued for some months. A year and a half later she had incomplete paraplegia and some rigidity; knee-jerks active and increased; ankle-clonus and plantar reflexes increased; R. V. = p. l.; L. V. = fingers before the face. There was optic atrophy in both eyes, more marked in the left. The above case is perhaps hardly worth adding to the list, except that it suggests the extension of an inflammatory condition from the optic nerve to the meninges.

I do not think that the public are sufficiently aware of the danger run in observing the sun. I take it that most children have in their time engaged in "sun-gazing competitions," although why there are not more cases of injury it is difficult to say. Possibly, however, some obscure cases of amblyopia may be due to this cause.

There is a case of central choroido-retinitis, with a drawing, quoted in Norris and Oliver's *System of Diseases of the Eye.*

(*January 31st*, 1901.)

Mr. Lawford said he had seen three cases of eclipse-blinding following the eclipse of May, 1900, but all of them belonged to the more ordinary category, and in none were there any except the slightest ophthalmoscopic changes. One was in a woman æt. 30 years, whom he saw twelve days after the eclipse. She had looked at the eclipse through a dull blue glass, and noticed a defect in both eyes imme-

diately afterwards. There was a relative but definite central scotoma for form, but not appreciably for colour. Vision was $\frac{6}{12}$ in each eye. Vision improved slowly, and when he last heard from the patient was practically normal. The second patient was a man æt. 26 years, who used a blue, and subsequently a smoked glass. He noticed the defect next day in the eye with which he looked at the eclipse—the right one, but was not seen until four and a half months later. No ophthalmoscopic change could be detected. He complained of the usual appearance of a disc over objects, which blurred everything he looked at. The third case was in a woman, æt. about 30 years, who studied the eclipse for a long time through ordinary smoked spectacles, using the left eye more than the right. The defect was chiefly in the left eye, and was noticed particularly about ten days afterwards.

All three cases had a slight central scotoma, and objects appeared blurred and distorted, but in no case were there any changes resembling those in Dr. Batten's patient. Nothing beyond rest to the eyes was prescribed.

Mr. Treacher Collins said he had seen two cases of defect of sight following prolonged exposure to the sun. The first case he had recorded in the *Royal London Ophthalmic Hospital Reports* (vol. xiv, p. 374). The patient was a schoolmistress æt. 49 years, who, on account of some coloured ring appearance around the sun, continued looking at it for five to ten minutes. When she turned away she saw two discs of a yellow colour before the objects she looked at. When he examined her some time afterwards, she had a central scotoma of the left eye, but the right had recovered completely. The outer part of the left disc was decidedly white. He saw her seven years after the exposure, and the disc remained white, and there was still the central scotoma. The other case occurred in connection with the eclipse last May. He saw the patient, a woman æt. 30, three weeks after the exposure. She had looked at the sun, without protection, for ten minutes, and then later on for another five minutes. For three days after-

wards everything looked purple; people's faces looked as though they were bruised; after that passed off the vision remained indistinct. Gradually it got better, and when she came to hospital she was able to see part of $\frac{6}{6}$ with the right eye, and $\frac{6}{12}$ with the left. Both fields for white were contracted, the left more than the right, and her fields for red and green were within a ten-degree circle. He had heard from her last October, and she said her sight was somewhat improved, but she had still defect in her left eye, at times something seemed to rotate in front of it. She also noticed distortion of objects; when she looked at a line it seemed to have a break in its continuity. The central letters of a line of print seemed larger than those at the sides. There were practically no ophthalmoscopic changes in either eye.

Mr. Bokenham said he had seen two such cases, one of them a slight one. One was in a poorly nourished girl of eighteen, who had looked at the eclipse with the right eye through a telescope shaded by a blue glass. Shortly afterwards she noticed a black spot surrounded by a bright margin. The symptoms passed off completely, though she had a small scotoma several days afterwards. The other case was much more serious. It occurred in an elderly Sister of Mercy, who had watched the eclipse through an ordinary blue glass. When he saw her she had several hæmorrhages on the nasal side of the retina, and a very marked large scotoma. There was much pain in the eye, and œdema of the disc. Vision had gone down to $\frac{5}{60}$. He kept her at complete rest and watched her, and her vision gradually improved; sight was now nearly $\frac{6}{12}$. There was still a well-marked scotoma, and the disc showed marked atrophic changes when last seen.

Mr. Jessop said he had published three cases of eclipse-blindness in the last number of the *St. Bartholomew's Hospital Reports*. The only treatment followed was eserine drops, and a bandage or peacock-green glasses to keep away the bright light. In the worst case of all he gave mercury. They all had marked diminution of vision as well as a positive scotoma. In two the vision

returned to $\frac{6}{6}$, in the third to $\frac{6}{12}$, but a central haziness in field of vision persisted in all. He would like to know if anyone had seen a case of eclipse-blindness in which the scotoma had completely disappeared.

Dr. G. A. Berry said he had seen a good many cases of blindness from the sun—eclipses and other causes, and had always found there was a permanent defect of vision in cases which had been very bad at the time; probably there were milder cases which did not so result. With regard to the remark of Dr. Batten as to the public being warned, he thought the public were aware of the danger. The general impression that there was safety in blue glasses was erroneous. It had been shown that the cause of light affecting the conjunctiva and retina was really the deleterious action of the ultra-violet rays. In Russia and other places it was the custom to wear red and yellowish spectacles, not blue. He thought ordinary smoked glasses were the safest.

Mr. Johnson Taylor asked whether there were any spots of keratitis punctata. The case was so different from others of eclipse-blindness that it was questionable whether the sun had anything to do with it. It might be a case of exudative choroido-retinitis, with obstruction of the retinal vessel merely as a coincidence.

Dr. Batten, in reply, said he had never detected keratitis punctata. He had fully considered the possibility that it was not due to the sun; but he examined the patient in every way, and could arrive at no other cause. The trouble was noticed on the evening of the day of the eclipse, and the œdema and opacities were progressive from that day. Nothing had since occurred to alter that view.

4. *A case of central choroiditis, simulating a neoplasm, watched for fourteen years.*

By Gustavus Hartridge.

I. C— came to the Royal Westminster Ophthalmic Hospital fourteen years ago for a failure of vision in the right eye.

R. V. $\frac{6}{24}$, eccentric.

L. V. $\frac{6}{6}$ and J. 1.

On examining the right eye one sees a large white patch about twice the size of the disc, reaching to the macular region, which it appeared to involve, and extending towards the temporal edge of the disc. The patch was distinctly raised, somewhat irregular on its surface, slightly pigmented in places, and showed new vessels on its surface. The patient seemed very healthy, and he gave a good family history. No history of syphilis.

The man has been seen, off and on, for fourteen years. Some nine months ago a convergent strabismus developed in his eye.

Two months ago numerous retinal hæmorrhages were seen, some of them extending well into the vitreous. On the patch was seen an irregular-shaped hæmorrrhage, which was not exactly on the patch itself, but in the vitreous just in front of it.

Examination of the urine.—Acid, sp. gr. 1010, alb. $\frac{1}{6}$. Three subsequent examinations have been made of the urine, and on two occasions albumen was present; on the other occasion he had taken some gin before coming to the hospital, and then the sp. gr. was 1000 and no albumen was found. I am of opinion that the affected area is slightly larger than it was when first seen, but I do not think it projects more.

(*Card specimen. March 14th*, 1901.)

5. *A case of choroiditis shown in an early stage in December*, 1898.*

By Gustavus Hartridge.

(With Plate III, fig. 1.)

Case of J. W. L—, shown, with a drawing, at the meeting of the Society on December 8th, 1898; the choroiditis was then in an early stage.

Now his vision is: R. V. $\frac{6}{6}$, Hm. ·50 D. = $\frac{6}{6}$; L. V. $\frac{6}{60}$.

Ophthalmoscopic examination.—Right disc still appears somewhat hyperæmic, with slightly blurred margin. Considerable pigmentary disturbance over the whole macular region, extending inwards to the edge of the disc. On the upper part of this disturbed area is a large white patch of choroidal atrophy, showing remains of deep layer of choroid.

Left disc.—Condition the same.

(*Card specimen. May 2nd*, 1901.)

The President said the case appeared similar to those of corneal nebulæ; they seemed as if they would not affect sight very much, but were often found very materially to do so. Ophthalmoscopically the eyes seemed practically the same, but vision in one was $\frac{6}{60}$, in the other $\frac{6}{6}$. Was any treatment adopted?

Mr. Hartridge said, in reply, that long-continued mercurial treatment was adopted. There was no history of syphilis.

* *Trans. Ophthalmological Society*, vol. xix, page 67.

6. *Nævoid condition of retina.*

By H. Work Dodd.

(With Plate III, fig. 2.)

In the case of Miss P—, a lady medical student, it may first be noticed that she shows a marked mixed nævus of the upper lip, in the middle portion. In the left fronto-temporal region there was formerly a nævus which was changed to its present cicatricial condition by vaccination in childhood. There are several large veins still to be seen in this region. In the left popliteal space there is another mixed nævus, and, finally, in the middle of the right upper lid, near the border, there is a very small one.

The retinal vessels in the left eye appear to be larger than those on the right side, and, although at first sight they seem to be irregular, yet when carefully examined, they will be found to keep broadly to normality in their entrances and courses. They divide rapidly, the arteries showing more branches than the veins. In some places the branches of both join their main trunks more at right angles than is usual. The vessels are markedly sinuous. The curves are almost all lateral, only in one or two places do they show a tendency to assume an antero-posterior character. In the big vessels the curves are large, becoming, as the vessels get less in size, small, in places assuming a sort of lateral ripple. There are no abnormal anastomoses. The dorsal white line is generally well marked in both arteries and veins; the yellow spot is perhaps more closely approached by vessels of larger size than usual. The disc shows a slight physiological cup, and there is some irregular pigmentation of its borders. There are also some opaque nerve-fibres to be seen. Two small congenital patches of pigment occur (not in the area of the plate), one below and to the outer side of the yellow spot, the other above and also to the temporal side of the fundus.

PLATE III.

Fig. 1 illustrates Mr. Gustavus Hartridge's case of Choroiditis (p. 81).

Fig. 2 illustrates Mr. H. Work Dodd's case of Nævoid Condition of the Retina (p. 82).

Fig. 1.

A. W. Head, *del.* Fig. 2. *Bale & Danielsson, Ltd., Chromo-Lith.*

There is a tiny white dot in the lower nasal segment of the retina.

The patient has several small cutaneous moles.

(*Card specimen. June 13th*, 1901.)

7. *Nævoid condition of the vessels of the disc, with nævus of the eyelid and face.*

By Gustavus Hartridge.

Miss M—, æt. 25 years, came to the Royal Westminster Ophthalmic Hospital for defective vision of the left eye. Patient noticed many years ago that the vision of the left eye was not so good as the right.

R. V. $\frac{6}{6}$, no Hm.; L. V. $\frac{6}{24}$, not improved.

Right, fundus normal; left, nævoid condition of the vessels on the disc, affecting chiefly the veins.

(*Card specimen. June 13th*, 1901.)

8. *Subretinal cystic swelling at centre of fundus.*

By J. Herbert Fisher.

Patient is a Jewess, æt. 25 years, married. Came to Moorfields Hospital on May 18th of this year, having discovered the left eye to be defective ten days before. The right has full vision, and its fundus and media are perfectly normal. The left can count fingers only.

The ophthalmoscope revealed a greyish spheroidal swelling nearly as large as the O. D. situated to the outer side of and somewhat above the level of the disc; it is globular, subretinal, and rather clearer at its centre than

its periphery; crossed by two retinal vessels which come forward nearly 5 D. upon it. It is surrounded by retinal change; up and in is a palisade of radiating spokes of retinal exudation and œdema; down and out the change is less regular, and consists of small white plaques less radiating in arrangement, and some retinal hæmorrhage. No movements of its wall have at any time been seen, and nothing in the nature of a scolex head.

Since first observed, the central cystic (?) swelling has distinctly decreased in size; by shrinking, the retinal vessels which crossed on its upper part are now left above the swelling altogether. On May 29th they crossed just on its upper margin, and are now left altogether above it.

The swelling is now not greater than one third diameter of O. D.; the retinal exudation has, on the other hand, become rather more extensive, and is seen to be less regular in pattern than when the ophthalmoscopic picture was made. (*Card specimen. June 13th*, 1901.)

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9. *Secondary carcinoma of both eyes following scirrhus of breast.*

By W. C. Rockliffe.

Margaret S—, æt. 46 years.

History.—The patient has been a powerful, hard-working, healthy woman all her life, with the exception of a scirrhus of right breast following an injury; removed October 20th, 1900. Married twenty-five years; no family; no miscarriages. Catamenia absent two years; bowels regular; no headache or sickness; urine 1020, no albumen or sugar; cardiac sounds normal. In March, 1901, sudden failure of vision in both eyes by artificial light. No pain, redness, or coloured rings.

April 16th, 1901.—First saw me at the Hull Royal Infirmary, complaining of mistiness of vision for near and distant objects by artificial or day light. Never worn spectacles. R. V. $\frac{6}{6}$ with + 2·5 D. $\frac{6}{6}$; reads 1 J. with difficulty with + 3·5 D. at about sixteen inches. F. of V. free. T. n. Pupil oval. General redness of O. D.; veins full; no neuritis. L. V.—F. at four feet with + ·5 D.; board only at 6 m. Pupil sluggish. T. n. Projection good. Intense optic neuritis with hæmorrhage into O. D. No cherry spot, but vessels very small at Y. S.; whole retina appears loose and detached at lower quadrant. Media clear.

Ordered three leeches to left temple; blister behind left ear. Pil. Hydrarg. Biniod. gr. $\frac{1}{3}$ t.d.

April 30th.—Ptyalised; fundus same, possibly more detachment. Ordered Pot. Iod. and Sodæ Salicyl.

May 28th.—P. L. only at extreme upper and outer field. Retina bulging into vitreous as four cysts.

June 4th.—No P. L.; 4 large, distinct, translucent cystic bulgings of detached retina compressing one another, and almost entirely filling two thirds of post. chamber. No cysticercus to be detected.

July 2nd.—*Left eye.*—Had acute neuralgic pain in the eye last week. Pupil rather larger than right. Consensual action only. Anterior chamber rather shallow. Lens clear. Slight external strabismus. Eyeball slightly congested—T + (?). Cysts not increased in size, but more opaque.

Right eye.—There is a general haziness of media, ? lens. The O. D. is pinker than usual, with ill-defined margins especially at upper and inner side. No hæmorrhages. The retina appears swollen over the greater part of the fundus. The arteries somewhat small.

R.V. $\frac{6}{36}$ with + 1·5 D. $\frac{6}{9}$; reads Jaeger No. 1 with great difficulty with + 2·5 D. Projection good. T. n.

The retinal vessels are tortuous, especially at the periphery, where there are choroidal changes of a doubtful character and a small detachment of the retina.

No enlargement of lymphatics.

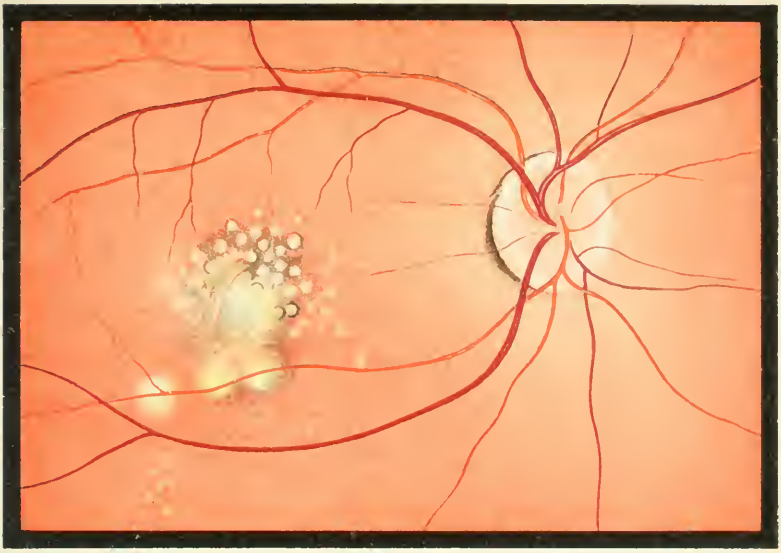
What is the nature and cause of the disease in each eye?

Mr. Devereux Marshall said the case corresponded in almost every particular with the recorded cases, about forty in number, where metastatic carcinoma of the eyeball had come on. In his paper in the *Ophthalmic Hospital Reports* he had looked up thirty cases, and they were in the eyes of twenty-four people, showing that several of the patients had both eyes affected, either together or one after the other. One of the cases, forming the basis of his paper, was seen by Mr. Lawford during life, and she had both eyes affected. The tension in most of these eyes was normal. He thought that was because carcinoma did not grow like sarcoma; it was not a large solid tumour growing into the globe, but generally flat, and occupying a considerable superficial area, so that there was not much disturbance of the intra-ocular fluids. In Mr. Rockliffe's case there was probably a layer of fluid just beneath the retina, causing a large amount of the detachment. He thought the right eye was becoming affected with the same condition, and, if left alone, doubtless other changes would take place. Excision of such eyes was not only justifiable, but was advisable, for if left alone the growth must go on increasing, and if the patient lived long enough, it would ulcerate, and probably cause a great deal of pain, whereas, if excised, the patient only lost a sightless eye. Unfortunately the right eye would probably become as bad as its fellow, but in the nterest of the patient excision seemed the better course to adopt. (*Card specimen. July 5th*, 1901.)

PLATE IV.

Fig. 1 illustrates Mr. R. Marcus Gunn's case of Recent Central Choroidal Disease of Unusual Appearance (p. 87).

Fig. 2 illustrates Mr. G. Brooksbank James's case of a Large Area of Retinal Pigmentation below the Optic Disc (p. 169).



M. H. Brooks, *del.*

Fig. 1.

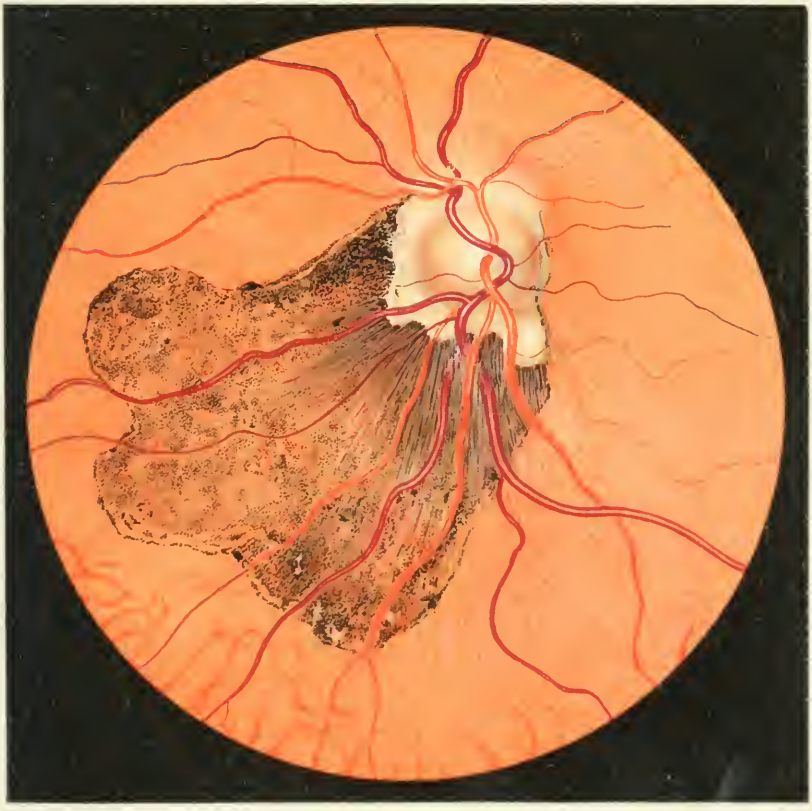


Fig. 2.

Bale & Danielsson, Ltd., Chromo-Lith

10. *Recent central choroidal disease of unusual appearance (? tuberculous).*

By R. Marcus Gunn.

(With Plate IV, fig. 1.)

Louisa L—, æt. 25 years, married. Came to Moorfields Eye Hospital on May 29th, 1901, complaining of visual failure in the right eye noticed three months previously.

R.V. = $\frac{6}{18}$, Hm. 0·75 not improved.

L.V. = $\frac{6}{6}$, no Hm.

Ophthalmoscopical examination.—Right eye media clear. At and near the macula there are one large and three smaller soft-edged patches, slightly elevated, of a pale yellowish hue. Around these patches, particularly above and inwards, are numerous small, pale, round spots; some of these are bordered by pigment, while others (further away from the larger patches) are not pigmented, and are seen with some difficulty; these latter are soft-edged.

Left eye normal.

There is no family or personal history of recognised importance. The patient has "slight dulness at the apex of the right lung, and breathing is comparatively harsh here, as if there might be a consolidation of old standing; but there is no evidence of any active change" (Dr. Comrie).

She was ordered complete eye rest, with dark glasses, and to take Syr. Ferr. Iodid. ʒss three times daily.

Since her first visit there have, I think, been several new small spots developed between the macula and the O. D. Her vision has improved considerably, and on last trial (June 29th) was $\frac{6}{9}$, well.

Note.—The feature of particular interest in this case is the association of small spots like those of "guttate choroiditis," with a recent exudative choroidal affection. The question is suggested whether this may not afford an

instance of the development of miliary tubercle around a central mass of caseating material.

(*Card specimen. July 5th*, 1901.)

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11. "*Hole*" *at the macula.*

By N. B. Harman, M.B.

The patient was a female, æt. 65 years, waistcoat maker, married, with healthy adult children. Applied at Middlesex Hospital for glasses to work with.

Right eye V. = $\frac{6}{60}$ with − 0·75 D. = $\frac{6}{36}$.
J. 14 with + 3 D. = J. 5.

Left eye V. = $\frac{6}{12}$ with −0·5 D. = $\frac{6}{9}$.
J. 4 with + 3 D. = J. 1.

The difference of vision was accounted for by the fundus appearance in the right eye. There was a small, oval, depressed area in the site of the macula; its size was about one third that of the disc, its edges were well defined, and it was surrounded by a ring of diffuse whiteness. The right field of vision showed a small central scotoma.

The case is like those reported by Mr. Treacher Collins and Mr. Ogilvie respectively in the Society's *Transactions* of 1900. In all these cases there was a history of the defect of vision following quickly upon a blow in the region of the eye. In this case the woman had had a severe blow forty-three years ago, but there is no history to connect this with any loss of visual power.

(*Card specimen. July 5th*, 1901.)

VII. PARASITES.

A case of intra-ocular hydatid.

By David J. Wood, M.D. (Capetown).

(With a postscript by Edward Nettleship.)

Jean C—, a cook about 30 years of age, was sent to me by the Cape Government Railways on April 6th, 1900. He had been acting as cook in a travelling saloon, and said that the great heat had "made his head bad" and damaged the sight in his right eye. His vision was $\frac{6}{24}$ in the right eye, $\frac{6}{5}$ in the left. After dilating his pupil, which was rather small, I observed fine vitreous opacities and an irregular track of choroido-retinal disturbance extending outwards from the disc. On looking at the extreme outer side I was surprised to find a pellucid, whitish, and beautifully rounded globe, with a denser whiteness in the centre. It seemed to be lying between the ora serrata and the ciliary region. The diagnosis was plain, though I had never before seen a hydatid in the eye. The patient had, it seems, been located on the line supplying the Kimberley relief force, and had drunk Modder river water for a long time.

On May 2nd, again dilated his pupil. His vision was no worse so far, and there was not, nor ever had been, any pain. On looking with the ophthalmoscope, I had the good fortune to see the remarkable spectacle of a living cysticercus hydatid in its cyst, clearly defined, and moving its suckers. Its head was against the anterior-inner surface of the cyst wall. It protruded its suckers slowly and regu-

larly, and pushed against the cyst as if trying to escape. Even the circles of hooklets were clearly visible, though the individual hooks were too small to be seen. No drawing could express the exceedingly delicate beauty of the cyst wall, nor convey an idea of the curious spectacle of a living and actively moving creature in the human eye.

I was only able to show the animal to one *confrère*, as it changed its position and became a mere white blur in the cyst within half an hour of my first seeing it.

After this I lost sight of the patient until the middle of June, as he was sent up country. His vision, he then said, was worse. I was unable to see the parasite properly through his small pupil.

On July 15th, I again dilated his pupil. His vision was reduced to perception of light only, the reason being an almost complete shallow, ripple-marked detachment of the retina. On the outer side I could no longer see the cyst, only a confused whiteness, though there seemed a denser centre where the embryo had been.

I believed this appearance to be due to inflammatory changes, and had just expressed this to a friend, when I caught sight of the hydatid lying loose in the lower part of the eye. One could see the margin of the cyst all round, so that it looked like a delicate soap bubble, only not iridescent. It had, as before, a dense white centre, whose long axis now was vertical. No details of the embryo were visible, but the cyst wall was very beautifully seen. It was evidently loose in the eye, and perhaps from this circumstance had caused the retinal detachment. I watched it some time but failed to see any movement.

Note by Edward Nettleship (August 7th).—The patient said he first noticed a "black spot" before the eye when on the Modder River about November last. The eye was quiet, with normal tension. Ophthalmoscopic examination (after a mydriatic) showed a very large, perfectly globular, translucent cyst at the inner and upper part of the fundus, so large that probably less than half its outline was in view at the same moment, and the extreme nasal, *i. e.* peripheral,

part of its circumference was too far forward to be in view. The edge of the circle was bright from total internal reflection, like the edge of a dislocated crystalline lens seen by focal light. The surface of the cyst was thickly covered by fine white grains seen with + 10 D. and higher, the so-called "calcareous granules" of the cyst wall. Rather below the centre of the cyst-circle, and evidently behind the cyst-wall, was a large ill-defined white opacity, which was especially dense at one end. On watching this part it showed at intervals of about 10 to 20 seconds a rather quick heaving movement, followed by a slower subsidence. At its *acme* the appearance was as if something opened, and a sharply defined little semicircle composed of tiny, short, radiating lines appeared for a second or less, and then subsided into the general white mist; evidently a part of the hook circle at the apex of the animal's "head."

The rest of the fundus was occupied by deep folds of detached retina varying in degree of opacity, but densely opaque at the nasal side; O.D. obscured by a fold, but retinal vessels traceable nearly up to it; no hæmorrhages seen. No opacities found in vitreous. The detachment slightly overlapped the extreme upper part of the cyst.

There was also sometimes a wave-like movement of the lower horizon of the cyst, always travelling in the same direction, viz. from the part of the cyst nearest the axis of the eye inwards towards its periphery at the nasal side of the fundus.

On the following evening several ophthalmic friends, as well as myself, examined the eye again. The "head" had gone further back and was less visible, and the movements were, on the whole, less active, possibly as the result of the effect of the mydriatic used (homatropine and cocaine).

I saw Jean C— again on September 7th; the worm was still moving much as before, but the "head" remained too deep to be definitely seen.

After each examination the patient had some hours of unpleasant heat and redness of the eye, and he said the inspections at Cape Town had caused the same symptoms.

It is necessary that he should have swallowed the egg of one of the two common species of tapeworm that live in the human intestine (*Tænia solium* and *Tænia mediocanellata*); and the probability is that he did so in drinking water fouled by fæcal matter containing the eggs from another person. I believe, however, it has been shown that a person with tapeworm may infect himself with cysticercus if he swallows the eggs of his own mature worm; the point is that the eggs must be acted upon in the stomach.

The patient told me he did not think he had had worms of any kind of late years, but he well remembered having more than one kind of worm when he was a lad. It is likely, therefore, that he had remained as careless about drinking-water and essential cleanliness in manhood as he had been when a youth. (*October* 18*th*, 1900.)

Dr. Little said he had seen several cases of cysticercus in the vitreous, but never one in the anterior chamber. Ophthalmoscopically the appearances were very similar to those in Dr. Wood's case. He had seen movement of the head and neck very distinctly in his examinations, and the whole body well defined; he had also observed the cyst change its position. He had watched such cases for three or four years. If left to themselves there was a slow process of choroido-retinitis, with very little external inflammation, but atrophy of the globe resulted, and the eye on account of pain or discomfort had to be removed. He had tried to extract the cyst in two cases but failed, and the attempt seemed to make matters worse. He believed the late von Graefe had successfully extracted one. In performing the operation he (Dr. Little) located the cyst as well as he could, and through an incision far back in the sclerotic he tried, with broad forceps, to fix some part of the animal; but nothing came of the attempt. He was now disposed to leave such cases alone. Interference seemed to hasten the onset of choroido-retinitis.

Mr. Nettleship said he did not know whether operation had been advised by Dr. Wood in the early stage or

not ; when he (Mr. Nettleship) saw the case in August the retina was detached and the eye useless. Would the eye have been more likely to remain quiet if operated on than if left alone ? He doubted it. Professor Zehender, in his Bowman Lecture some years ago, gave an account of the German position in regard to it. He gathered that the results of extraction of the cysticercus by operation were not in the main encouraging. It was interesting to hear from Dr. Little that if the eye were left alone it usually shrank and became troublesome after some years.

Postscript (*July* 31*st*, 1901) *by Dr. David J. Wood.*

Retina completely detached. No perception of light. Cyst can be just made out amongst the folds of the retina ; it seems no smaller than formerly. No pain or discomfort other than occasional flashes of light.

VIII. DISEASES OF THE ORBIT AND SINUSES.

1. *Double frontal sinus mischief simulating symmetrical exostoses in the neighbourhood of the lacrymal sac.*

By John Griffith.

R. G. A—, æt. 15 years, was sent to me by Dr. Burney in September last as a case of unusual interest.

A tumour is present on each side of the nose. The one on the left side was first noticed four years ago; it slowly increased in size for two years, and then ceased growing. That on the right side is only of eight months' duration, and he believes it to be still on the increase.

A tumour can be felt in the neighbourhood of each lacrymal sac. The left one is the larger, and has perceptibly displaced the eyeball outwards. It is hard and craggy, and reminds one of an ivory exostosis, except at one point where fluctuation can be felt. The one on the right side has a soft elastic (? fluctuating) area over the firm osseous base. This soft covering was regarded as a distended lacrymal sac, but both nasal ducts are free, so that water can be freely syringed through without any resistance. The distance from the centre of the nose to the right inner canthus is 2·5 cm., and to the left inner canthus 3·25 cm. The left pupil is displaced outwards 7·5 mm. further than the right. Epiphora is present from displacement of the puncta. There is no interference with the ocular movements; he possesses binocular vision, and in no position of regard can diplopia be elicited. The optic discs are normal. Uncorrected R. V. $= \frac{6}{9}$; L. V. $\frac{6}{24}$; both eyes astigmatic.

The nasal cavities have not been examined; there is no nasal obstruction, nor any nasal discharge.

There is a history of scarlet fever, which the boy attributes as the cause.

(*Card specimen. October 18th*, 1900).

(I am inclined to agree with the members who regard it as a case of bilateral distension of the frontal sinus.)

2. *Case of probable frontal mucocele.*

By Walter H. H. Jessop.

Louisa P—, æt. 10 years.

In March, 1899, whilst playing, she struck her left eye with a ruler, and for some days after there was ocular pain and profuse lacrymation. The pain ceased, but the lacrymation continued, and in May, 1899, the mother noticed a swelling on the inner part of the upper eyelid about the size of a grain of corn, and took her to the West Ham Hospital. The swelling gradually increased in size, and during the last eight months has increased much more rapidly. She has complained of frontal headache almost constantly since last June. When five years old, whilst recovering from scarlet fever, she fell down and struck her nose on the end of the bed. There was much bleeding from the left nostril, which was plugged regularly every night for some weeks. For some months after this there was a "bad smell" from the nose.

Family history good. The child has had scarlet fever, measles, and chicken-pox. She is a healthy-looking, rosy-cheeked girl; general health good: throat and ears normal.

Left eye.—The eyeball is slightly proptosed and displaced downwards and outwards; the movements of the eyeball are good. The line of the upper eyelid curves downwards and outwards, and is displaced by the swelling; the lower

lid is at a lower level than the right. Just above the inner canthus and internal tarsal ligament is an oval swelling beneath the skin, which is freely movable over it. The swelling is about three quarters of an inch long, elastic, fluctuating, and diminishing very little in size from pressure. At its upper margin the bone is much thickened and nodulated; there is also a bony edge to be felt below.

Left eye.—The tunics and media normal; V. $\frac{6}{12}$.

Right eye.—Normal except for astigmatism; V. $\frac{6}{12}$.

Mr. Walsham examined the nose and reported:—The middle turbinal bone was swollen on the left side. The olfactory slit was natural. There was no sign of any pus from the infundibulum, nor could a probe be passed into it. No enlargement of ethmoidal bulla; naso-frontal probe could not be passed. Nothing abnormal in nose.

(*Card specimen. January 31st*, 1901.)

Mr. A. P. Wells said he had a case which terminated fatally last year in a boy 14 to 15 years of age, in which there was necrosis. The acuteness of the condition necessitated two operations. He came first with one eye and then with the other displaced downwards. It was a simple matter to open the sinus and let out the material by cutting just inside the inner margin of the orbit. But when associated with necrosis, a very unpleasant percentage died by infection of the diplöe and subsequent cerebral affections, as in the case of the boy he alluded to. He had been told by two experienced rhinologists that they had had similar cases, and foreign authors also made mention of them. The incision he had mentioned left no conspicuous scar, and in some cases the drain by the infundibulum into the nose could be established without any difficulty. If the material were not let out by a puncture he thought it would shortly make its own way out, as it was not far from perforation, and by feeling one could get a good idea of how thin the bone had become.

Mr. Tatham Thompson said he had a similar case under care at the present time. When first seen about a year

ago, there was great proptosis. At that time the tumour was very hard and indurated, so that at first he thought it was an exostosis, and therefore kept the patient under observation. The bone was thinned, so that he could almost feel fluctuation, and that led him to think he was dealing with a mucocele. A colleague opened it two months ago, inserted a small drainage-tube, and all the proptosis subsided. There were no bad symptoms. It was still discharging to some extent, but the general health was good. There was no family history of tuberculous trouble.

Dr. Bronner suggested that it was possibly a dermoid cyst. He had under his care a child of twelve with a diseased frontal sinus from scarlet fever. He thought the modern treatment of frontal sinus disease was not dangerous. The present case was not serious, because there was no pus.

Dr. Little thought the case was one of mucocele or disease of the frontal sinus. He had seen cases in subjects quite as young as this, and he thought if Mr. Jessop were to make a bold opening and drain it through the nose, he would get a satisfactory result.

Dr. Berry thought there could be no doubt that the modern operation of cutting down on to an empyema in the frontal sinus along the eyebrow was the best treatment. But in many cases there seemed to be a difficulty in being quite sure of the diagnosis, even when in consultation with men who had made a specialty of the nose, throat, and ear. Therefore, when there was a distinct swelling externally, it was better to make an exploratory puncture, and be sure it was a frontal sinus. If so, and a swelling had appeared externally, a passage could be found in the frontal sinus, and then the more radical operation could be proceeded with.

IX. INJURIES OF THE EYE AND ORBIT.

1. *Rupture of the lamina cribrosa and optic nerve-fibres at the papilla.*

Exhibited by W. Lang.

(With Plate V, figs. 1 and 2.)

Ernest D—, æt. 14 years, was struck on September 9th at 5.30 p.m., when playing with another boy, by a clothes prop on the right eye. He vomited several times during the night. Early the following morning he came to Moorfields looking very ill, and complaining of pain in the right eye. The blow appeared to have been received on the lower lid, a little to the nasal side of its centre; both lids were swollen and ecchymosed. The conjunctiva was ecchymosed and torn slightly near the outer side. The cornea, iris, pupil, and lens were normal. T.n. With the ophthalmoscope, the disc, or rather the scleral ring, seemed to be divided in two by a sharply-defined horizontal line, which separated the upper two thirds from the lower third. The latter had the appearance of a coloboma of the optic nerve-sheath, being dark, but its depth could not be measured. The upper part consisted of the pink nerve-head with its surface directed downwards and forwards, its lower border being drawn backwards. The superior retinal vessels coursed over the scleral ring in a normal manner, but the inferior ones ended at the lower margin of the ring, where they were partly buried in a large retinal hæmorrhage which existed at the lower part of the fundus. The upper part of the field of vision (*see chart*) was deficient, the limitation corresponding to what one would expect from the injury. V. $= \frac{6}{60}$ (with a correcting glass).

PLATE V.

Figs. 1 and 2 illustrate Mr. William Lang's case of Rupture of the Lamina Cribrosa and Optic Nerve-fibres at the Papilla (p. 98).

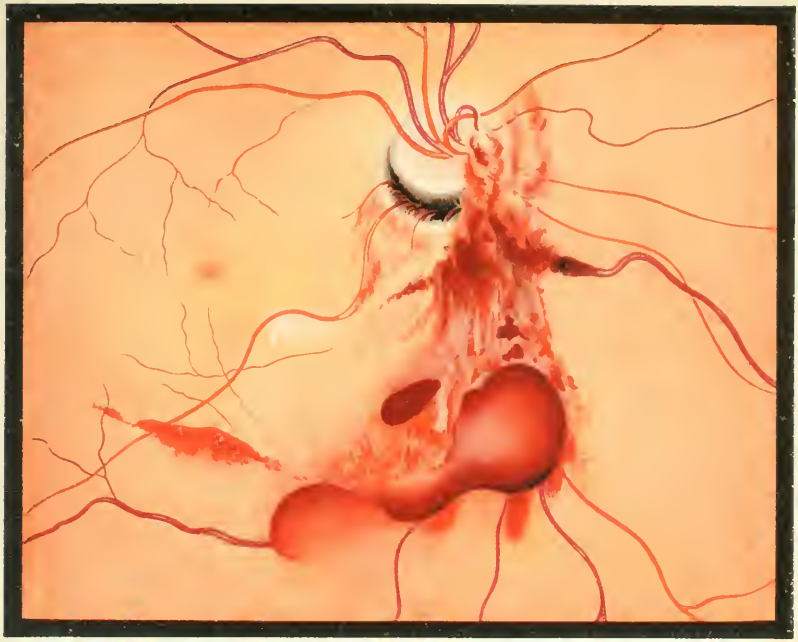
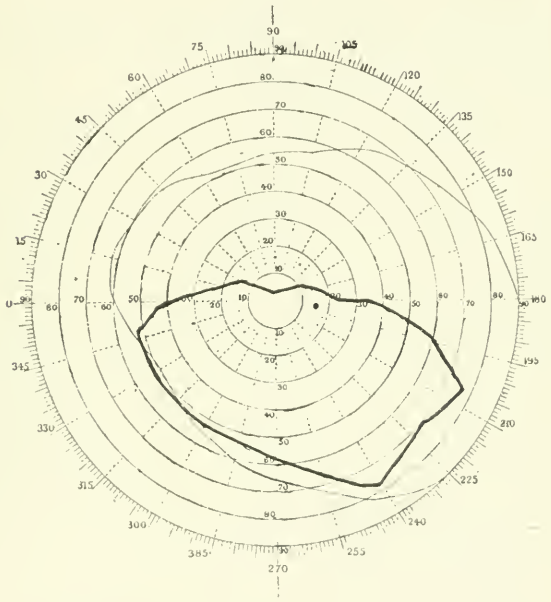


Fig. 1.

M. H. Brooks, *del.* Fig. 2. *Bale & Danielsson, Ltd., Chromo-Lith.*

The patient was taken into the hospital and the accompanying drawing made the next day. Since then changes have steadily gone on, until at present the chasm in the optic nerve-sheath has filled in, the dark cavern being represented by a shallow white pit 1·5 D. in depth. Most of the blood-clot has become decolourised and is represented by a white mass in the lower part of the fundus, whose surface is seen with a + 12 D. It tails off

Fig. 2.



Ernest D—. Field of vision of the right (injured) eye.

towards the equator of the globe and ends in a feathery margin, which is separated from the retinal vessels beneath by 2 D. to 3 D. Parallactic movements between the two can be obtained. Between the outer part of the disc and the y. s. region, the retina contains white patches, where hæmorrhage has taken place. The field and the vision remain unaltered.

(*Card specimen. October 18th*, 1900.)

July 18th, 1901.—Vision unaltered. Field slightly more contracted. White exudation streams directly forwards into vitreous. Slight pigmentary disturbance in choroid below disc. All remains of blood gone. The inner part of the cleft visible as a black crescent, remainder covered by white exudation (see Fig. 2).

2. *Diminished tension and the appearances of optic neuritis persisting for nine months after a wound of the orbit by a shot from an air-gun.*

By E. Treacher Collins and J. S. Hinnell, M.D.

Horace W—, æt. 16 years, on January 1st was shot in the right eye by an air-gun pellet at fourteen yards. His eye bled a little, became very painful, and the sight became blurred.

He was seen by Dr. Hinnell two days later, who found V. of R. E. = counting fingers; no wound in conjunctiva or lids, but on inner side of lower lid, at junction of inner and middle thirds, a raised granulation one eighth of an inch in diameter. The globe was generally congested, but no wound could be discovered; T. — 3; cornea and anterior chamber normal; streak of blood on lens capsule. Lens and vitreous clear.

The congestion gradually became less, but the tension remained low. About two weeks after the injury, optic neuritis was first noted, but it may have been present sooner. The swelling of the optic disc was estimated at 9 D. No hæmorrhage seen, and no other changes in the fundus at that time.

The patient was not seen again for some time. On June 5th Dr. Hinnell found one dark moving vitreous opacity down and in, and down and out streaks and patches of pigment, and a small patch of choroidal atrophy. There was no rupture of the choroid and no detachment

of the retina. The patient came to the Moorfields Hospital first on July 16th, 1900. The granulation on the inner surface of the lower lid was still to be seen, also a streak of blood at the lower pupillary margin. There was a small opacity in the lower part of the lens, and some floating opacities in the vitreous. The tension was — 2. V. = $\frac{1}{60}$. Field much contracted at upper and inner part. The optic disc had the appearance of intense neuritis. The disc itself and the fundus for some distance around it, were best seen with a + 7 D. glass. The retinal vessels near the disc were in part obscured. The veins were large, the arteries small. The pigmentation in the lower and outer part of the fundus was extensive, some of the patches branching like those seen in retinitis pigmentosa. There were some white dots in the lower part of the fundus, and some white lines running along the line of the retinal vessels in the upper part. Left eye, healthy; V. = $\frac{6}{9}$, and J. 1.

Examination with the X rays by Dr. Mackenzie Davidson on July 31st, showed the presence of a bullet, situated so far forwards that Dr. Davidson thought it must be visible clinically, and he found that after the patient hung his head forwards it came into view beneath the conjunctiva, in the fornix at the outer canthus. On August 2nd, after several attempts, with the patient bending forwards, the bullet was removed. It was so loose beneath the conjunctiva, that each time an attempt was made to grasp it, it slipped back out of sight; at last, however, it was successfully secured.

The patient left the hospital on August 8th, with the condition of the eye itself unchanged. T. still — 2.

October 18th.—V. of R. = $\frac{2}{60}$, T. — 2. The appearances of optic neuritis are still present; the disc and a large part of the rest of the fundus are best seen with a + 7 D. or + 8 D.

(*Card specimen. October 18th*, 1900.)

3. *Cases of blindness from gunshot injury of the orbit.*

By E. Nettleship.

In the cases referred to, the sight of one eye, sometimes of both eyes, was lost or injured by the passage of a bullet through the orbit, without wound of the eyeball. I have said through the orbit as a general statement; but in two of my cases there was some doubt whether the projectile actually penetrated into the orbital cavity, and in one of these the missile was a piece of shell, not a bullet. I am excluding cases in which the damage involved only the ocular muscles or their motor nerves, or the fifth nerve, and those of an altogether different kind, in which more or less loss of vision has followed injury to some part of the visual path or centre within the skull.

The cases I shall mention are such as have been described as "indirect gunshot injuries of the eye," in which the passage of a bullet through some part of the orbit is followed immediately by free bleeding into the vitreous, and as this clears up, by various and mixed appearances of choroidal rupture, choroido-retinal exudation, and changes at the disc; there is no softening or shrinking of the globe, nor any signs, from beginning to end, that the sclerotic has been either ruptured or cut through. Granting the correctness of this diagnosis the question is, how are the violent intra-ocular changes produced? This is answered for one case by Waldeyer's full examination of an eye removed by Cohn, the main particulars of which are as follows:

Cohn's* patient, a soldier, æt. 23 years, wounded by a Chassepot bullet at outer border of L. orbit on October 30th, 1870. Unconscious fifteen minutes, and then found he could not see light with L. eye. Bullet and a piece of bone removed in half an hour. Seen by Cohn December 2nd (four and a half weeks); wound in lids, etc., healed.

* Hermann Cohn, *Schussverletzungen des Auges*, Erlangen, 1872, Case 28, with coloured plate and woodcut.

L. eye doubtful p. l., much opacity in vitreous; a large, dimly seen pale area at fundus, a few retinal vessels visible, choroidal blood-patches and lines like exposed sclerotic seen at periphery. R. eye normal. In January, 1871, L. vitreous clearer, and in March (six months) almost clear. At both dates the centre of fundus showed (*a*) a large area of dense white opacity with pigmented border and radiating processes, neither vessels nor optic disc being visible upon it (? exudation or exposed sclerotic): (*b*) a smaller white patch with hæmorrhage at its edge, and very tortuous retinal vessels on its surface: (*c*) hæmorrhage and pigment spots between *b* and periphery. The R. having now become irritable, Cohn excised L. Professor Waldeyer found the globe of normal size and shape. The large area at fundus consisted of a layer of white tissue, in places as much as 1·5 mm. thick, containing scattered pigment, and representing retina and choroid, these structures being indistinguishably fused together at the centre of the patch but reappearing, with alterations, at its circumference. Vitreous at this part fibrous and adherent. Sclerotic corresponding to thickest part of exudation, slightly thickened (not a muscular insertion), but showing no other change except a deposit of blood and small cells on its outer surface. The other spot (*b*) at the fundus was also an exudation. Cohn concludes that a contusion which directly affects only the outer surface of the sclerotic, may set up severe and extensive plastic inflammation in the subjacent choroid, retina, and vitreous.

Cohn gives other cases (Nos. 22, 25, 26) in his paper of gross changes, such as vitreous hæmorrhage, rupture of choroid, atrophy of optic nerve, and detachment of retina from bullet wounds of the orbit without wound of the eye; but his case 28 (given above) is the only one in which the eyeball was examined anatomically.

In 1879 Oettingen * wrote on the same subject from

* G. v. Oettingen, *Die Indirecten Lasionen des Auges bei Schussverletzungen der Orbitalgegend*, Stuttgart, 1879.

cases seen in the Russo-Turkish war of 1877–8, and concluded that gunshot injuries of the orbit that do not directly implicate the eye may cause rupture of choroid, detachment of retina, and hæmorrhage into the vitreous, and that later on plastic choroido-retinitis and optic neuritis often arise; (such changes occurred, more or less, in thirty of Oettingen's forty cases.) He diagnosed laceration of the optic nerve in one or two cases where the blindness was instant, total, and permanent. He thinks there are but few cases in which permanent blindness should, with our present knowledge, be attributed to mere concussion of the retina or optic nerve.

A very instructive case, in which retinal changes limited to the yellow-spot region were caused by a bullet that lodged close to the postero-internal part of the eyeball, was published by Mr. Monteith Ogilvie in the last volume of our *Transactions*.*

A case in which lesions at the back of the fundus followed the entry of small shots into the orbit without evidence of penetration of the eyeball was published by Mr. Lawford in 1882.†

My own cases are as follows:

Case 1.‡—*Rupture of choroid and probably retina, and atrophy of the optic disc, from bullet-wound of lower-outer part of orbit. Communication between a retinal and choroidal vein.*

Captain D— (53, 23), æt. 32 years. On October 20th, 1899, wound of left orbit by Mauser bullet at very short range ("fifty yards"); entrance at lower-outer part, notching malar bone a little external to line of infra-orbital canal, exit just over (*i. e.* superficial to) articulation of lower jaw, below the zygoma, with gutter-wound of lower part of external ear in continuation of the bullet's path.

* *Ophth. Soc. Trans.*, vol. xx, p. 198.

† Lawford, ibid., vol. ii, p. 15.

‡ This case was mentioned in my remarks on Mr. Ogilvie's paper, ibid., vol. xx, p. 201.

Inability to open the eyelids, at first from swelling, afterwards from want of power, also stiffness of lower jaw for a time. As soon as eyelids could be opened, about two months after injury, eye found to be "blind." When seen six months after injury (April, 1900), still slight left ptosis and slight defect of outward rotation of globe; eye a very little less prominent than right. No anæsthesia. Smell perfect. A deep notch in lower outer rim of orbit, and small scar over it, and another scar over articulation of jaw. T. of eyeball slightly less than R; pupil acts a little to direct light, and well to mydriatic; p.l. only at outer part of F.; O. D. pale all over; no detachment of retina; retinal vessels (except those mentioned below) natural. A large area of disorganised choroid and retina elongated from before backwards, extending from lower outer part of equator up to and including y. s., and consisting of exposed sclerotic, disturbance of adjacent choroid, fibrous bands, and splits in the choroid diverging from the main tract, these changes ceasing rather gradually, both towards periphery and y.s. The lower temporal retinal artery very small, and cannot be traced after it reaches the atrophic area; its companion vein also entirely lost for a space between O. D. and the area of disease, but reappears on distal side of this area, as if it had formed a new connection with the choroidal system at the peripheral border of the rupture.

The appearances were those of an extensive rupture of choroid and retina on the side nearest to the bullet track; yet the line of wound was such that the bullet could not anywhere in its course have been less than about 6 mm. from the outer surface of the sclerotic. The almost total blindness was not explained by the choroido-retinal changes, and we must assume that the optic nerve was destroyed either by immediate hæmorrhage into its substance, by vibration injury, or by subsequent inflammation extending from the damaged region of choroid; as no examination of sight was made until several weeks after the injury the point cannot be decided.

Case 2.—*Indirect contusion injuries of choroid and vitreous with some optic neuritis, from shell wound of superior maxillary region.*

Captain W— (53, 115), æt. 29 years, extensive wound of right side of face by fragment of shell, February 24th, 1900, followed in a fortnight by erysipelas and some secondary hæmorrhage; later, good healing after loss of fragments of bone. Condition three months later, when I saw him, end of May, 1900:—Disfigurement of right upper maxillary region, nose, and upper lip; floor of orbit quite regular; no displacement of eyeball. Sight of right eye very defective (18 Jaeger, but only when he looked a little below the object); relative defect of entire upper half of F., but no absolute loss, "a hazy veil across the top of the sight;" p. sluggish to direct light and larger than the other; T. n.; no sign of wound of globe; position and movements of eye normal; refraction about 2 D. of M. at y. s., and he was aware the eye had been short-sighted for many years, but sight had been otherwise perfect till the wound. The loss of sight was not noticed till, he thinks, three weeks after the injury, but he was in no condition to observe it in the earlier period. In lower part of vitreous I found many large whitish masses between which the retinal and choroidal vessels were seen, and looked normal; several narrow lines of choroidal rupture passed from just below O. D., up and out, reaching beyond y. s., and between these ruptures the pigment epithelium was collected into stippled dots; O. D. hazy and probably rather swollen (seen clearly without lens), arteries rather small, veins normal; no detachment. L. eye $\frac{6}{5}$ and all normal.

November 9th, 1900.—Vitreous now quite clear; disturbance of pigment epithelium over whole lower part of fundus as at y. s.; choroidal ruptures as before. V. slightly improved.

The damage to the choroid here was approximately opposite, not contiguous, to the seat of impact, for the vibration presumably reached the anterior lower part of the

eyeball through the bone forming the orbital floor. The intra-ocular changes differed from those of ordinary rupture of the choroid by direct violence chiefly in the larger amount of opacity of the vitreous. The appearance of some papillitis is not extremely rare after rupture of the choroid.

Case 3.—*Rupture of choroid and retina, followed by exudation from passage of bullet behind eyeball; communication between retinal and choroidal vessels on the ruptured area; partial atrophy of optic nerve; ptosis, strabismus.*

N— (33, 80), æt. 24 years, shot himself in the right temple with a revolver loaded with a small round bullet and a very small charge of powder; no loss of consciousness and no serious results except the damage to the eye and orbit. Extreme swelling of eyelids and protrusion of eye, and when this subsided ptosis and strabismus with considerable loss of sight of R. eye. Bullet not found.

I saw him in January, 1895, five months after the injury. There was a small scar in right temple three quarters of an inch behind outer end of eyebrow, a little behind and level with junction of frontal and malar bones. There was semi-ptosis, divergence, and limitation of upward movement, and some proptosis. Some doubtful fulness at upper inner part of orbit. P. varying under direct light from 6 mm. to 3·5 mm. V. $\frac{6}{60}$ with a gap in F. which included fixation point. Media clear; at upper central part of fundus a large oblong area of epithelial disturbance and denudation passing from just above O. D. out and down, so as to include y. s.; at centre of this area a triradiate patch of grey fibrous tissue, into which a large retinal artery and vein plunge with marked tortuosities and disappear; O. D. pale (greyish) and sharply defined. L. eye $\frac{6}{6}$, all normal. No loss of smell in either nostril.

Mr. W. T. Lister, who saw this gentleman three years after the above examination, kindly writes to me that the conditions are unaltered, and particularly that the levator palpebræ and superior rectus have not recovered. Mr.

Lister found some opacities in the vitreous which I had presumably overlooked.

Case 4.—*Bullet passing behind both eyes from right to left; total blindness of right with hæmorrhage into vitreous, other conditions being obscured; damage to fifth and motor nerves. Damage to left optic nerve.*

Joseph W— (S.W. ii, 77), a young man, shot himself with a bullet from a pistol held close to right temple October 5th, 1888. Brought into St. Thomas's Hospital unconscious, but regained consciousness in half an hour, when it was found that with the R. eye he had no p. l., and with L. he could see hand, both ps. being dilated and fixed. A small wound blackened by powder one inch behind right external angular process and half an inch above right zygoma; bullet could be felt under skin immediately below left zygoma. Bullet extracted at once; no bone removed; drainage-tube passed quite through head, but removed after one day. Great swelling of R. lids and conjunctiva, but not much of L.

October 31st (two and a half weeks), Mr. Lawford examined the eyes and found right fundus almost entirely darkened by blood in vitreous; no p. l.; cornea anæsthetic and movements of eye much impaired, swelling nearly gone. L. media clear; O. D. doubtfully pale.

I saw him in November and again in December, and found the R. still absolutely blind, p. motionless, cornea quite anæsthetic; slight ptosis, lateral movements abolished, upward movement impaired, downward movement nearly full; lower part of fundus opaque and grey, reflex from upper part good and some retinal vessels seen there; no evidence of detachment; state of choroid could not be made out. L., some defect of upward movement; p. large, acting a little and very slowly to light; no anæsthesia of cornea; O. D. decidedly pale; no other changes. V. with +2 D. $\frac{6}{18}$, F. (to 15 mm. white object) shows slight general contraction.

CASE 5.—*Bullet passing behind both eyes from right to left, and probably remaining in L. orbit; immediate total blindness of both eyes with profuse bleeding into vitreous and damage to choroid; optic nerves probably cut across close to eyeball.*

B— (29, 116), æt. 32 years, shot himself with a bullet from revolver held about an inch from right temple in October, 1893. He never saw light after that moment. He did not lose consciousness, but in a few minutes stood up and, although absolutely blind, managed to find his way to the house of a friend about three hundred yards off. In bed a fortnight with some headache on right side, swelling of eyelids and loss of power in the upper lids, so that he had to raise them mechanically with his finger, but no other symptoms. Bullet not found.

I saw him in December, just two months after the occurrence, and found the eyes externally healthy. They were deeply set in the orbits, but I thought this was natural, not due to wasting of orbital tissues from the injury. T. n. in each eye; no p. l. with either eye; smell quite, and taste almost, abolished since the injury; no anæsthesia of fifth nerve; hearing good; no ptosis, but upward movement of both eyes almost abolished, downward movements of L. defective; lateral movements full but accompanied by oscillation of each eye when turned inwards. Pupils wide: R. 8 mm.; L. 6·5 mm. A small bluish scar a short distance behind right external angular process. Since about a month after the injury he had become aware at times, especially if he nodded or shook his head, of a "rattling or splashing" sound in the left forehead; I heard it easily on placing my ear near his forehead; no pain accompanied the sound. The ophthalmoscope showed in each eye a few small opacities in the lens, the deep layers of the vitreous opaque, with streaks and moving grey clouds most dense in lower part, fundus reflex fairly good in upper part, and some large areas of atrophied choroid

could be made out there; in R. eye also a large area of red blood with fringed edges: neither O.D.s nor retinal vessels were visible; no evidence of retinal detachment.

I saw him again six months later (eight months after the injury). The L. had in the interval suffered an attack of inflammation, with severe pain lasting some weeks, but had quieted again; the rattling noise in left forehead could no longer be heard.

CASE 6.—*Bullet wound at inner side of orbit causing severe damage of choroid and retina, chiefly on opposite side of eye, with atrophy of optic disc, and ptosis.*

S— (4, 85), æt. 19 years, wounded by bullet fired from distance of four feet, and entering a little to left side of root of nose. Bullet not found. Much swelling of left eyelids followed by permanent drooping of upper lid; sight much damaged from the beginning, but got still worse afterwards. Seen, with Mr. Johnson Smith, on May 28th, 1880, three months after injury. A depression in bone beneath scar; left palpebral fissure only half as wide as right, and lids rather hollow, as if from some wasting of orbital tissues; has p. l. in lower-outer part of F. only; direct reflex action of pupil very slight; indirect reflex and associated action full; movements of eye good. Extensive and severe changes in lower-outer parts of choroid and retina around O. D., passing down and out, including y. s., to periphery, and consisting of large areas of pigment accumulation with fibrous tissue spurs, gradually thinning off at periphery into mere epithelial disturbance with pigmentation of retina; O. D. pale and rather hazy; upper main retinal vein covered by a mass of pigment close to O. D.; the corresponding artery emerged from upper edge of O. D., but whether this apparent communication with choroidal system was natural or the result of the injury could not be decided; arteries on O. D. pulsated very easily on pressure; refraction slightly H. R. eye $\frac{6}{6}$ and all normal.

Remarks.—In Case 4 there is little doubt that the absolute loss of sight was caused by complete severance of the optic nerve, and in Case 5 of both optic nerves, by the bullet in its passage across the orbit just behind the eye. But the feature of chief interest to us this evening in the entire group is the extensive damage of choroid and retina, without wound of eyeball; the optic nerve may be severed or otherwise injured at the same time, but the lesions of choroid and retina are independent of injury to the nerve. In one of the most typical (Case 1), where the optic nerve was not cut across, although the choroido-retinal changes were on the side of the eye nearest to the path of the Mauser bullet, the bullet, as already mentioned, could not anywhere have been less than 6 mm. from the sclerotic; whilst in Cases 3, 4 and 5, though the bullet was probably nearer to the globe, and may even have grazed its outer surface, there is no reason to suppose that the sclerotic was cut through by the projectile. In none of my cases was there any reason to think the eye had been wounded by splinters of bone.

These indirect lesions are, it seems, to be explained by assuming that, at any rate, in the case of the soft tissues which during life are almost fluid, the force of the projectile is partly changed into vibrations of the tissue particles, which vibrations radiate in lines "from the long axis of the bullet track so forcibly that," as Professor Stevenson,* of Netley, puts it, "they act as secondary missiles." We must suppose that such lines of intense vibration, impinging on the outer surface of the eyeball, though unable to cause disintegration of the dense sclerotic, can pass through it with sufficient force to produce rupture of the much less firmly knit choroid and retina immediately beneath. It is also probable that dragging on a muscular insertion by the bullet may produce an effect on the subjacent parts.

That such radiating vibrations of an incompressible fluid are able to cause violent solutions of continuity in

* Surgeon-Colonel W. F. Stevenson, *Wounds in War*, 1897, p. 69.

neighbouring solids, has been shown experimentally by Coler (quoted by Stevenson), and his experiments have been repeated and verified by Stevenson.* If a vessel made of sheet lead, shaped like a jampot, left open at the top and containing nothing but air, be fired at, the bullet makes a clean entrance and exit wound without distorting the outline of the vessel. But if the vessel be filled with water, the top still being left open, the effect of a bullet at very high velocity is to rend the vessel to pieces.† The experiments were made with a modern military rifle at, for it, very short range. I have made a few trials of the same kind with a much feebler weapon, namely, a rook rifle carrying a small leaden bullet, and only sighted up to 150 yards, and have obtained similar, although much slighter, results. The effect of this bullet, fired at three feet (1 m.), at a cylindrical vessel of thin sheet lead,‡ seven inches high by four inches diameter, and full of water, is to cause an expansion of the wall of the cylinder which, at the level of the bullet track, increases its circumference by rather more than one inch, with a gradual diminution above and below that plane; the wound of exit is also larger and less regular than in the case of the empty vessel. When the muzzle of the gun was close to the vessel—only one inch (25 mm.) off—the amount of expansion was rather greater, and there was a circular depression surrounding the entrance wound, due in part, I suppose, to the pressure of the exploded gases. A similar open vessel, containing only air, was not altered in shape at all when wounded at the same distances (three feet and one inch); also the exit wound was small and circular, and there was no depression of the area of entrance.

Vibration lesions are, I believe, well known to occur in other parts than the eye, and they are, I suppose, included in the so-called "explosive" results of certain bullet wounds; and it appears that the vibrations may, under some conditions, pass harmlessly through a dense structure,

* Stevenson, loc. cit., p. 63.

† For illustrations see Stevenson's work, p. 61, etc.

‡ Weighing 3 lbs. a square foot.

even through bone, and yet cause destructive changes in a soft tissue, such as the spinal cord, beyond it, as we have already seen to occur in the case of the sclerotic and choroid.

The explanation just offered seems to have taken the place of other theories, such as that of the pad of compressed air in front of the bullet, and that of hydraulic pressure.

Returning to the intra-ocular changes:—the amount of hæmorrhage into the vitreous appears to vary greatly. In such of my cases as were seen early the bleeding was largest when there was reason to believe that the optic nerve had been torn across close to the eye (Cases 3, 4, and 5). In these cases there was coincident extensive contusion—or vibration—injury of the choroid at its most vascular part, the posterior pole, and we probably need not seek further than this for the source of the bleeding. Indeed Aschmann has published two cases the first of which shows that the optic nerve may be lacerated close to the globe and the retina torn from the optic disc without the occurrence of much intra-ocular hæmorrhage unless the choroid be damaged also. In his first case a deep wound of the orbit through the lower lid by a stick caused immediate blindness without change of ocular tension; three days later the media were found to be clear, the retina torn from O. D. and detached from the choroid by effused blood; the place of the O. D. grey and much excavated, giving the appearance of a hole in the sclerotic; no mention of rupture of choroid or of subsequent increase of intra-ocular hæmorrhage. The eye was excised for suppuration in the orbit, requiring exploration, between two and three weeks after the injury; there was partial laceration of optic nerve at or close to the point of entrance of arteria centralis, the torn part being necrotic and the surrounding parts of the nerve much inflamed and infiltrated with blood, the retinal nerve-fibres completely torn from the disc at the level of the lamina cribrosa, the latter structure considerably retracted, and the place of the disc filled with granulation tissue. This

patient died under the anæsthetic, and a fissure fracture of the roof of the orbit with subdural pus and extensive meningitis were found; the case had been obscured by an eruption diagnosed as measles about the eighth day.* In Aschmann's other case† the choroid was torn and the bleeding excessive; orbit penetrated through lower lid by a thrust from the end of an umbrella; three days later examination showed the place of the disc taken by a large hæmorrhage, extensive detachment of retina and retinal hæmorrhages, and several (obscurely seen) choroidal ruptures; a few days later the globe filled with blood, and no further examination was possible. Two months after injury, eye removed for continued pain; the globe was ruptured at the back and quite full of blood; the optic or retinal nerve-fibres torn across close to the lamina cribrosa, the disc replaced by granulation tissue, the choroid detached by blood from the sclerotic, and the retina from the choroid; no note of the ocular tension in this case.

It is interesting to compare the course of events in the cases of bullet wound with what follows optico-ciliary neurotomy. Although the extra-ocular disturbance is violent and extensive in both, the area affected in neurotomy is larger but less defined than in the bullet wound; and neurotomy is not performed clinically upon healthy eyes. We are, therefore, prepared to find that the intra-ocular changes are not identical in the two groups.

Experimental section of the optic nerve and posterior ciliary nerves and vessels of healthy eyes in rabbits was found by Poncet‡ to be followed from the eighth day onwards by degeneration of the nerve elements of the retina with increase of its fibrous structures, marked papillitis, and some inflammation on the outer surface of the vitreous. The re-establishment of retinal circulation, of which he

* Aschmann, *Wunden des Sehnerven*, Inaug. Diss., Zurich, 1884, Case 20, with two figures.

† Ibid., Case 21.

‡ Poncet, Landolt's *Arch d'Ophtalmologie*, t. i, pp. 120—145, January, 1881.

found evidence by the eighth day, was too late to save the essential retinal structures. Later on the choroid near the disc became atrophied, and adhesions and capillary vascular connections formed between it and the retina. Finally the degenerated retina became pigmented. Poncet says very little about hæmorrhage into the eye. He states that the traumatic inflammation behind the eye scarcely invaded the sclerotic at all, and it will be remembered that Waldeyer made a similar observation in Cohn's specimen of indirect bullet injury. Berlin's* somewhat earlier experiments of the same kind had given similar results as regards the final pigmentation of the retina.

The results of experimental section of the optic nerve and posterior ciliary vessels, as stated by Berlin and Poncet, agree closely with those in a case diagnosed clinically as wound of optic nerve with laceration of the central vessels by Pagenstecher† in 1869. A penetrating wound of the orbit by an iron bar caused permanent blindness of the corresponding eye, with the following series of changes:— on the third or fourth day, dense white opacity of retina at the posterior part of the fundus, with emptiness of almost every vessel, and white spots and empty vessels in the choroid; about the eighteenth day or earlier, collateral circulation at border of O. D., filling of retinal vessels, clearing of retinal haze, restoration of choroidal circulation, but commencing pigment formation; finally, disappearance of peri-papillary anastomoses, shrinking of retinal vessels, atrophy of patches of choroid and formation of pigment in the retina, as well as increase of previous pigment patches in choroid.

The liability to adhesive inflammation of retina and choroid, both after experimental neurotomy and indirect gunshot injury, may account for the comparative rarity of retinal detachment in the latter group. Detachment was not found in any of my six cases, though it could not be

* Berlin, *Klin. Monatsbl. f. Augenheilk.*, 1871, p. 278. Abstract in Nagel's *Jahresbericht*, 1871, p. 328.

† Pagenstecher, *Graefe's Arch.*, 1869, p. 223.

excluded, owing to the state of the vitreous, in Cases 4 and 5. But the healthy state of the eye before the injury doubtless takes a large share in preserving it alike from retinal detachment and extensive subchoroidal hæmorrhage.

The absence or, at most, slight degree, of enophthalmos is also of interest, considering the amount of bleeding that occurs in the loose tissues of the orbit in many of the cases.

The relation in place between the bullet track and the intra-ocular lesions was not always the same. Whenever the projectile unmistakably traversed the orbit (Cases 1, 3, 4, 5), the damage was at the part of the fundus nearest to the wound track. In the two cases (Nos. 2 and 6) where there was some doubt whether the orbit was actually entered by the projectile, the fundus changes were at a part remote from what seemed to be the wound path; and this may be compared with the similar relation between the part struck and the part damaged, in ordinary rupture of the choroid from a blow on the front of the eye by a blunt body at low velocity.

It is, of course, to be expected that orbital nerves other than the optic will be injured in many of the cases, especially when the bullet has passed behind the eyeball. In Case 5, where both optic nerves were cut across, there was loss of smell; and in the similar case, No. 4, anæsthesia of one cornea. More or less ophthalmoplegia was present in five of the six cases, the exception (Case 2) being one of those in which invasion of the orbit by the projectile was not proved (shell wound of face). The commonest paralytic result was ptosis (every case except No. 2), incomplete when the case came under my notice, but stated to have been complete for a time in the early periods. Next in frequency, in at least three cases (Nos. 4, 5, and 6), was loss of upward movement; decided loss of lateral movement was seen in only one case (No. 4).

(*October 18th*, 1900.)

The PRESIDENT said that if any members had had personal experience of similar cases, the Society would be glad to hear them. The only case he had had he alluded to at a previous meeting. It was that of an officer who was shot, the bullet entering at the fronto-parietal suture, passing downwards through the anterior lobe of the brain, emerging through the roof of his mouth, and finishing up by taking two teeth out of the lower jaw on the opposite side. Here there was no choroidal change, but the disc was atrophic, and the eye was practically blind. One interesting point noted by Mr. Nettleship was that in these cases the injury in the choroid and retina was in the immediate track of the bullets as far as could be ascertained.

Mr. HARTRIDGE said he saw, a month ago, a soldier recently returned from Africa, who had been shot at Modder River, the bullet having entered the upper part of his forehead, coming out at the external canthus of the left eye, quite close to the margin of the lids, but without touching the bone. The man's eye appeared perfectly normal, and it was very difficult to see the scar of entrance and exit. The eye movements were perfect, but the man said he could not see out of the eye. He had a very large pigmented area in the choroid; he had scarcely more than perception of light. The optic nerve was a little pale, but not distinctly white; the retinal vessels were fairly normal. There was no rupture of choroid. The man told him that the day after the injury Mr. Cargill saw him in South Africa, and said at that time that he had a very large choroidal hæmorrhage. When he (Mr. Hartridge) saw the case there was no sign of hæmorrhage, and the vitreous was perfectly clear. The bullet must just have grazed the posterior pole of the eyeball. It certainly had not interfered with the muscles. He was shot with a Mauser bullet, and got quite well in ten days, although the bullet must have gone through the anterior portion of his brain.

Dr. GRUBER said he had met with a case similar to the last described, one of attempted suicide. All that could be seen was extensive pigmentation of the macula; he

thought that in such cases the posterior ciliary arteries are injured.

Mr. Donald Gunn said he recently heard of a case which it might be worth Mr. Nettleship's while following up, to see if the later changes were of the nature described. Dr. Wood, of Capetown, said he had recently seen a patient who had had a bullet wound of his left orbit. The bullet struck him on the right inner canthus, crossed part of the nose, traversed the left orbit, and came out at the left temple. As far as he could recollect, the right eye had escaped injury with the exception of a small abrasion of the cornea. The left eye was then full of blood; he could not say whether it showed fundus changes later. The bullet was from a Lee-Metford rifle.

Mr. Treacher Collins had seen two cases like those described by Mr. Nettleship.

Case 1.—Corporal C—, æt. 29 years, was on February 8th, 1900, at the battle of Paardeberg; he ran short of ammunition, and raised himself to procure some more; he at once received a Mauser bullet through his head. It entered on the right side just above the zygomatic arch, midway between the ear and the outer margin of the orbit. It passed out on the left side through the frontal bone just above the external angular process. He did not lose consciousness, but on being hit raised himself a little higher, and at once got another bullet through his shoulder. His lower jaw became fixed, and for about seven weeks, it had to be forced open to admit food. He said the sight of his left eye went within the course of about twenty-four hours. The lids of his right eye became much swollen, and he does not know how soon after the injury that eye went blind. When seen on August 9th, 1900, six months after the injury, he had some ptosis of the right upper lid, he could only raise it just above the centre of the cornea. There was defective movement of both eyes upwards and some restriction in the movement of the right eye outwards. There was a nebula at the lower part of the left cornea; both pupils were dilated and inactive, and he had no per-

ception of light with either eye. Ophthalmoscopically the optic disc in the right eye was seen to be grey and the retinal arteries small; on the outer side of the optic disc was a considerable area of choroidal atrophy with extensive pigmentation of the retina. In the left eye, at the upper part of the fundus and covering the optic disc, was a white mass with a granular-looking surface, which extended upwards as high as could be seen, and which ended below with a well-defined margin, from beneath which the retinal vessels were seen to appear. There were also some superficial areas of choroidal atrophy visible, and some floating opacities in the vitreous. He (Mr. Collins) took it that this patient must have had a wound of the right optic nerve, either directly from the bullet or indirectly from fractured bone, and that probably one of the posterior ciliary arteries on the outer side of the optic nerve had been cut across, which accounted for the area of choroidal atrophy and the pigmentation on the outer side of the disc. In the left eye there had probably been some hæmorrhage into the vitreous chamber, and the white mass which was seen was decolourised blood-clot.

Case 2.—Mr. X—, æt. 34 years, was a melancholic patient, deaf and dumb after scarlet fever. In July, 1898, he attempted suicide by shooting; the bullet entered the right temple, and came out at the left cheek below the left orbit. When seen three months later he was blind and in a most miserable and pitiable condition; the only way in which he could be communicated with was by the sense of touch, and his only pleasure in life came through the sense of taste. The right eyeball was shrunken and puckered; there was an opacity at the lower margin of the cornea, which was small and puckered. The left eyeball was displaced backwards in the orbit (enophthalmos) and also drawn upwards. Its tension was normal, and he had bare perception of light, but projection was defective. The pupil was dilated and acted slightly to light. The cornea and lens were clear; by reflected light only a grey reflex could be obtained, except upwards and

outwards, where a dull red reflex was seen. He (Mr. Collins) thought that in this case the right eye must have been perforated by the bullet at the posterior part, and that in the left eye there must have been an extensive hæmorrhage into the vitreous, brought about in the way Mr. Nettleship had suggested.

Mr. J. Tatham Thompson.—Corporal C—, of the 1st Welsh Regiment, was wounded at Paardeberg; Mauser bullet entered just about half an inch from the outer canthus of left eye, passed slightly down and back, and came out close to the right external auditory meatus. The wound of entrance is represented by a mere dimple, that of exit by a stellate scar. The vision of the left eye is *nil*, marked atrophy of disc, and a great deal of scarring of choroid. Whether the sclera and choroid were actually seared by the bullet, or whether this is inflammatory I cannot say; I rather think both. The movements are quite normal.

Right eye is quite unaffected; $\frac{6}{6}$ reads J. 1; fundus normal.

I heard from my staff nurse, who is at the front, last week of another case where the bullet entered at the left side, and is lying under the right masseter muscle. Vision of left eye *nil*, right eye perception of movement.

Mr. Nettleship, in reply, said he was glad to find that his cases were not the only ones observed. He suggested that full particulars of the cases mentioned should be furnished to the Society in connection with the paper. The one point which he had not referred to, because he had no information about it in his cases, was the immediate effect of the injury on the tension. In his cases tension was normal, or in one or two perhaps a little *minus*. The case shown to-night by Mr. Collins with persistently low tension, and Mr. Ogilvie's case, where an eye with very low tension was excised soon after the injury and no rupture of sclerotic found, both belonged to the group of indirect lesions under consideration. It was very interesting if violent vibration injuries producing intra-

ocular hæmorrhage could also cause marked decrease of tension, either temporary or permanent. If that were so, he supposed it was due to some effect of the injury upon the secretion of intra-ocular fluid. It was of practical interest to know that in a case in which the tension was found to be *minus* shortly after a violent indirect injury of the eye, we need not now assume that that lowered tension necessarily meant wound or rupture of the globe.

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4. *Case of bullet wound of orbit followed by blindness of both eyes.*

By E. Treacher Collins and A. Vernon Ford.

Albert B—, æt. 23 years, a seaman, was on June 21st, 1900, while in China with Admiral Seymour's relief expedition for Pekin, hit on the head with a bullet. He was unconscious for a month afterwards; when he recovered he found he was quite blind, and has remained so ever since. He has had several attacks of bleeding from both nose and mouth. Until about seven or eight weeks ago he was unable to open the left eye.

There is a scar one and a half inches long on his right cheek, below the margin of the right orbit, and running down and out from the side of the nose. There is a second scar, circular in shape, situated on the left side of the head, two inches above the zygomatic arch, and midway between the outer margin of the orbit and the external ear.

Examination with the X rays (by Mr. Mackenzie Davidson) shows no trace of any bullet. He has no perception

of light with either eye. Both pupils are widely dilated and inactive. Tension of each eye is normal. The movements of the right eye are normal. There is some lacrymal obstruction and distension of the lacrymal sac on the right side. The left eyeball is displaced backwards in the orbit (enophthalmos); its movements in all directions are restricted, mostly upwards and downwards There is slight ptosis.

Ophthalmoscopic appearances.—Right eye.—The optic disc is of a greyish-white colour. The retinal arteries are small, those running down and in being reduced to white lines.

There is a rupture of the choroid below the disc, situated about a disc's breadth from it, and a second rupture running concentric with the other, but nearer the periphery, down and in. At the margin of each rupture there is a considerable amount of pigmentation.

Left eye.—The optic disc is hidden by a white mass which extends upwards and inwards as far as can be seen. At the margins of this white mass, elsewhere, the retina is detached. There are some white lines in the detached retina, behind the retinal vessels, down and out. On the surface of the white mass is seen an irregularly shaped, bright-coloured patch of blood.

(*Card specimen. November 8th*, 1900.)

5. *Concussion injuries of the left eyeball.*

By J. B. Lawford.

H. W. C—, æt. 13 years.

Injury by a slug from an air-gun about December 29th, 1900. No perforation of globe.

Ophthalmoscopic appearances.—Hole at macula roughly circular, sharply defined; coarsely pigmented floor. *Detachment of retina* showing a *rent* in the retina at temporal periphery. Deposit of pigment along the vessels on the detached portion. Much fine opacity in the vitreous, and several large strands of opacity. Vision = counting fingers at 50 cm. Field defective in nasal part.

(*Card specimen. January 31st*, 1901.)

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6. *A case of bullet injury of the occipital lobes, with loss of the lower half of each visual field.*

By Anderson Critchett.

During the campaign in South Africa, Captain A—, while pursuing De Wet, was struck in the occipital region by a bullet, probably a Lee-Metford, on July 24th, 1900. The captain, who at the time was standing up, was in the act of addressing his men who were on his left. He fell on being struck, but there was an almost entire absence of pain, and he likened the blow to that from a racquet ball. He at once lost his sight, but did not become unconscious till about half an hour later. He remained in a comatose condition for about eight days, and when he returned to consciousness he was still blind. The first dawn of returning sight was noticed on the fifteenth day after the receipt of the injury, when the patient was able to distinguish the flash of a lighted match. The vision slowly but steadily improved from that date.

Two semilunar scars, the result of operative interference, are present in the scalp; and within the area of each a small scar, indicative of the bullet wound, can be made out.

He was trephined on the day following the injury, he believes at both the point of entry and exit of the bullet.

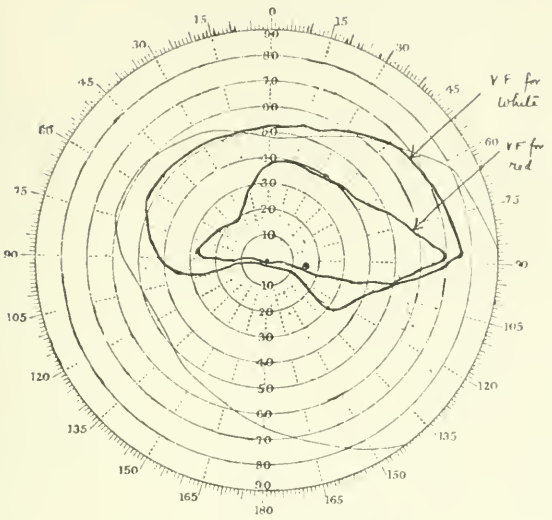
The trephine aperture at the seat of entry, where there is a distinct depression, is situated two and a quarter inches above Reid's base line on the right side, and in a vertical line upwards from the posterior border of the base of the right mastoid process. It is one and a half inches below the parietal eminence, and slightly behind it, four inches from the external occipital protuberance.

The aperture of exit is much less clearly defined than that of entry, but there exists a depression within the area of the semilunar scar two inches above the external occipital protuberance, half an inch to the left of the middle line, and about one inch below the lambda. The pupils act directly and consensually; the fundi are normal; and the vision of each eye $= \frac{6}{6}$ and J. 1. The patient has colour-blindness, being unable to recognise green, which he calls pink. On this account he was unable to enter the navy.

He has lost the greater part of the lower half of each visual field, the line of limitation being irregular (*see charts*). This feature affords the chief point of interest in the case, as it is evidence of interference with the cortical centre for vision. The scalp scars of the bullet wounds are on the same level, therefore the bullet must have traversed the skull horizontally from before backwards, and from right to left. There can be little doubt that the brain was damaged, as the captain was informed by a trustworthy witness that brain-substance was found upon his sleeve.

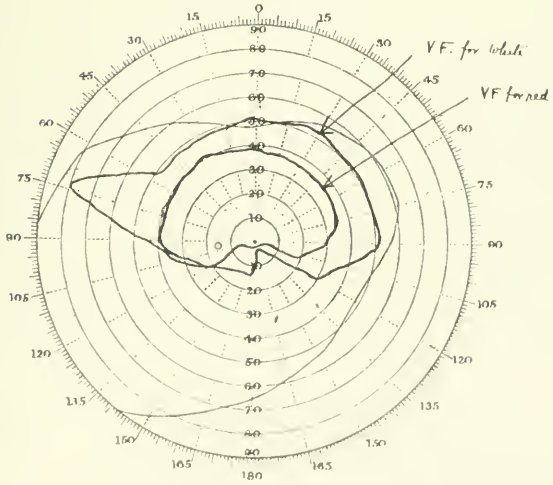
From the position of the scars and trephine holes, it appears that the course of the bullet must have been through the anterior part of the right middle occipital convolution, and, as it passed backwards, it must have penetrated the right cuneus, and injured also the left cuneus at the apex of the left occipital lobe, and quitted the skull one and a half inches above and slightly to the left of the torcular Herophili.

FIG. 3.



Field of vision of the right eye.

FIG. 3 *a*.



Field of vision of the left eye.

The sudden failure of vision immediately on the receipt of the bullet wound suggests destruction of the cortical centre for sight. It is, however, evident that total destruction could not have occurred, as the vision has, to a large extent, returned. The sudden impact probably caused a temporary paralysis of the visual area from concussion. That there was a considerable extravasation of blood is proved by the history of the rapid onset of unconsciousness. It is impossible to estimate in what degree the present loss of the visual fields was caused by the extravasation of blood, or by the actual injury by the bullet. If the latter be assumed to have produced the permanent failure of the lower half of the visual fields, it is difficult to explain the exact symmetry of the visual loss. So far as I am aware, no identical case has yet been recorded.

Since the above was written, a somewhat similar, and very interesting, case has come under my notice, published in a recent number of the *Lancet* by my friend and colleague, Mr. Herbert Page. The case is one of injury in the right occipital region, attended with homonymous hemianopsia on the left side. It was complete when the fields were first taken, but, subsequently, after a period of about five months, the visual fields were almost restored to their natural limits. Mr. Page's case resembles mine in that there was an injury in the occipital region causing damage to the cortical centre for sight. The lesion in his case, however, involved the cortical centre on one side only, and completely for a time; whereas the damage in the case I record must have involved both sides, and only partially, producing what appears to be a bilateral quadrantic hemianopsia. In neither Mr. Page's case nor my own was optic neuritis seen. (*January 31st*, 1901.)

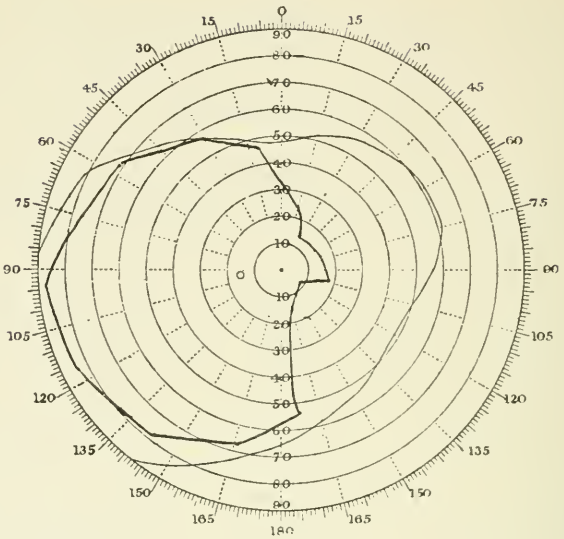
7. *Bullet wound of occipital region.*

By J. Herbert Fisher.

W. S—, late 3rd Grenadier Guards, now of Corps of Commissionaires, seen November 23rd, 1900. Received a bullet wound at back of head, at Belmont, November 23rd, 1899. Was five days unconscious, and on recovering consciousness was in Wynberg Hospital; he found that he had no perception of light, and was unable to speak to anyone. Mr. Makins had operated on him, and, I learn, removed the depressed bone of a gutter fracture; much brain matter came away, both at time of operation and subsequently, the dura mater being opened and lacerated. Mr. Makins records pupils as large, but acting normally during the period of blindness. The recovery of vision was gradual; in a day or two after recovering his senses light began to come back, "like day dawning," and a fortnight later he was able to read a word or two. At the present time he thinks the vision is as good in one eye as in the other. I find now, twelve months after his wound, R. V. $= \frac{5}{12}$, $-$ 0·5 D. $= \frac{5}{6}$; L. $\frac{5}{9}$ accurately, $-$ 0·5 D. $= \frac{5}{5}$ well. In neither eye, however, is he really myopic. Pupils are equal and active to light; no suggestion of hemiopic pupil. Movements are quite full; no diplopia; says he has no difficulty with colours.

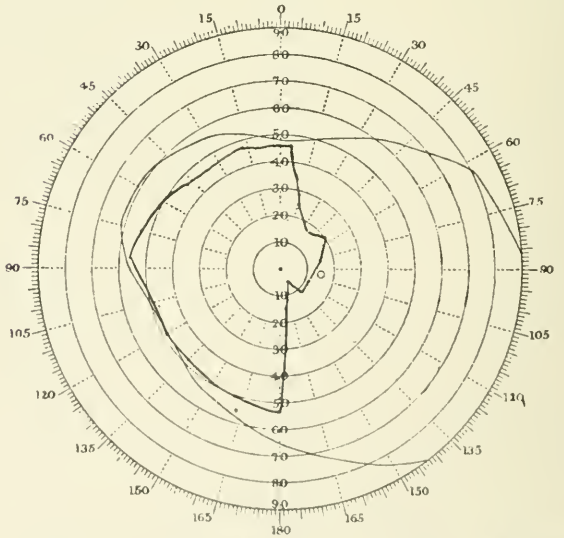
His fields are as attached: his answers to the perimeter test are ready, reliable, and given with military precision; has quite characteristic right homonymous hemiopia; it comes out equally well to the hand test; he is aware of his loss of power to see on his right, and runs into obstacles on this side. He has now a broad, straight, linear scar in the scalp, with loss of bone underneath, and imparted cerebral pulsation. The scar is about 2—3 inches long, begins about one inch to the left of midline over the occipital bone, about the level of the external

FIG. 4.



W. S—. Left field of vision for white spot 10 mm.; taken in good daylight.

FIG. 5.



W. S—. Right field of vision for white spot 10 mm.; taken in good daylight.

occipital protuberance, and runs upwards and to the right, terminating in the upper part of the occipital table on the right side, not far from the lambdoid suture. The scar is so tender to the touch that he can scarcely wear his uniform cap. Recovery of speech is even now not perfect; it appears, however, to be a difficulty of articulation, and not of the nature of word-blindness.

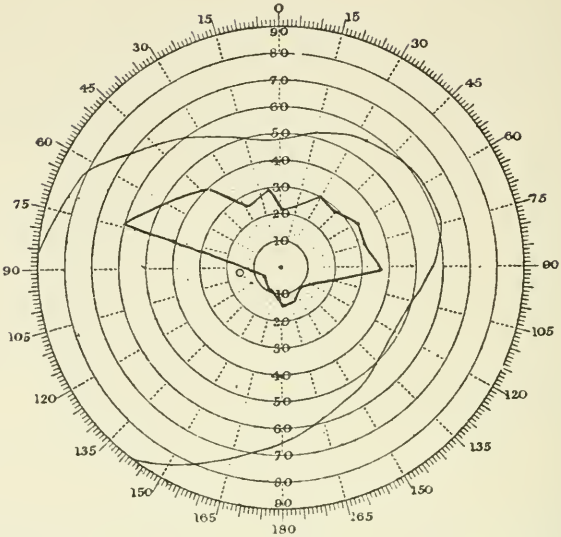
Swanzy, in his *Handbook*, says, "Aphasia occasionally accompanies *right cortical* hemianopsia, although it is not easy to offer a satisfactory explanation of this fact." The same authority, in his article in Norris and Oliver's *System* again refers to this association of symptoms, but it appears to be to mind- and word-blindness rather than to motor aphasia (difficulty of articulation) that he alludes.

The lesion is one of the left occipital cortex in the cuneate lobe and neighbourhood of the calcarine fissure.

Mr. Makins has rough notes of another case of right hemianopsia with aphasic symptoms, but has not been able to trace the patient since his return to this country.

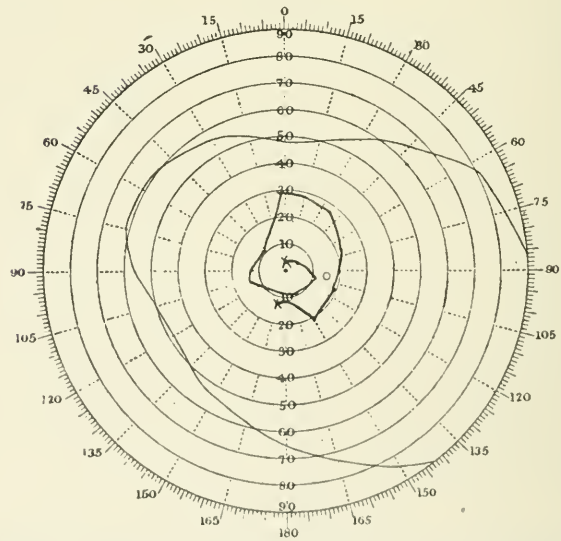
J. W. R—, æt. 21¾ years, late Argyll and Sutherland Highlanders, was wounded at Magersfontein at 500 yards range, December 11th, 1899, and was seen by me December 1st, 1900. My notes are as follows:—"Blind for two days after his wound; sight gradually returned. Mr. Makins thinks the bullet did not perforate but grazed the occiput, causing a depressed fracture. There was an operation for bone depression under Mr. Makins' direction soon after the wound. About a week later again symptoms of brain irritation from pressure necessitated another operation, when more bone was removed. Some fragments were situated as deeply as 2 inches from the surface. Both operations were at Orange River; he was laid up at Wynberg till January 31st, 1900, and reached home February 26th, 1900. Is now an assistant postman at Stirling, N.B. He has now a lozenge-shaped area of loss of bone at the apex of the occipital table. The sides of the lozenge measure from ½ inch to ¾ inch. The depressed area involves both sides

FIG. 6.



J. W. R—. Left field of vision for white spot 10 mm.; taken in good artificial light.

FIG. 6*a*.



J. W. R—. Right field of vision for white spot 10 mm.; taken in good artificial light.

of the mid-line, but rather more on the right than the left side.

"Says his vision is defective, is apt to run up against people, was nearly knocked down by a vehicle in Glasgow, and is accused by his friends of passing them in the street. He volunteers the statement that when he looks at me with the left eye he sees the upper but not the lower part of my face. Has been seen by an ophthalmic surgeon in Glasgow, who said his vision incapacitated him from further service, and gave a certificate to this effect. He seems satisfied with his present position, and at first viewed my motives with suspicion; but on gaining his confidence I found that the vision of R. or L. $\bar{c}$ + 3·0 D. sph. was $\frac{5}{6}$, and together $\bar{c}$ + 3·0 D. s. in front of each $\frac{5}{5}$ partly. Fundi were normal. The pupils equal, active, and normal.

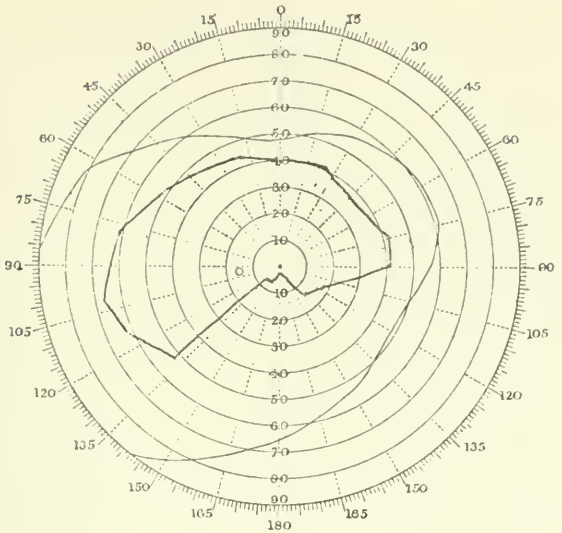
"The fields as attached. Left, taken first, is definite and reliable in showing the lower half of the field to be lost, although it may perhaps be smaller than it really should be. The right shows the spiral functional field, and this element in it obscures anything else. The lower half may or may not be lost. He does not say he is aware of the loss of the lower half of the sight. With both eyes open he guides himself in my room without difficulty or mistake, and watching him away from my front door on a dull afternoon he walks away briskly and with no difficulty; is in no anxiety about getting about London, with which he is unfamiliar. Certainly, the field of the right must be bigger than the chart shows. I am satisfied as to the loss of the lower half of the left field. On Henschen's view as to the upper part of the visual centres in the occipital cortex representing the upper dorsal retinal quadrants, the lower part the lower quadrants, and the bottom of the calcarine fissure the maculæ, the state is explicable, assuming the lower half of the right field to be lost, which may quite probably be the case. Mr. Marcus Gunn, in his Bowman Lecture, accepts this view of the visual centres. J. W. R— has a bone lesion high up, situated at the apex of the occipital table, so that it would be likely to be accompanied by

damage to the upper part of both cuneate lobes above the calcarine fissure, while the lower parts escaped; the fracture being fairly median might be expected to damage both half-centres in their upper portions."

My notes also say J. W. R— enlisted as a drummer-boy when æt. 14 years. By estimation he has hypermetropia of 6·0 D. in each; refraction, of course, was not measured when he enlisted, only acuteness of vision taken. His hypermetropia would be likely to tell more on his unaided direct vision now, especially after his injury, etc.

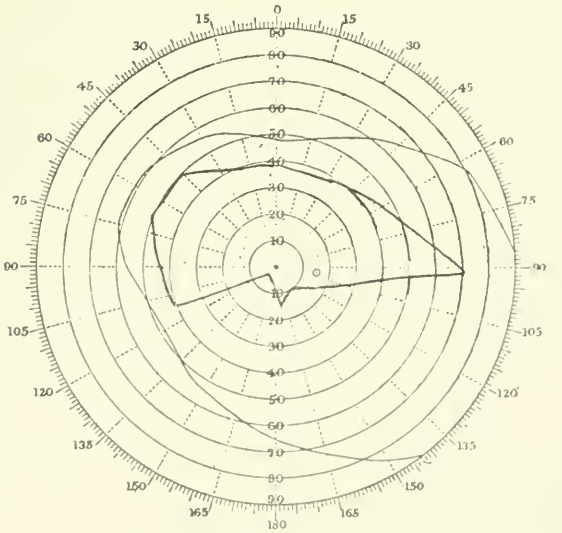
Private W—, 2nd Seaforth Highlanders, æt. 26 years, sent to me by Mr. G. H. Makins, December 8th, 1900. Says he was wounded by a Mauser bullet at Spitzkop, Orange River Colony, May 25th, 1900, range within 1000 yards. Remained semi-conscious for five days; on coming to himself all was dark. Six days later he first recovered perception of light; gradual improvement in vision has taken place since, but thinks it stationary for the last month. Mr. Makins says he was quite blind when brought in. Had right hemiplegia and right facial paralysis, but no aphasia; the pupils were equal, of moderate size, but inactive to light. He has two independent injuries to the skull; one is a small depressed wound the size of the tip of the little finger, pulsating, with loss of bone, and situated just above the line of the right lateral sinus, *i. e.* above the tentorium cerebelli, about halfway between the external occipital protuberance and a vertical line drawn through the right external auditory meatus. The other is a gutter depressed fracture, which had been much comminuted, in the upper part of the left parietal bone; it is about 3½ inches long, begins in the sagittal suture 1½ inches in front of the apex of the occipital bone, and runs a curved course bowed ontwards to terminate in a line drawn vertically through the left external auditory meatus about 1½ to 2 inches from the sagittal suture. I made out no pulsation in the site of this fracture. Scars are not tender. No operations were performed on this patient.

FIG. 7.



Private W—. Left field of vision for white spot 10 mm.; taken in good artificial light.

FIG. 7 *a*.



Private W—. Right field of vision for white spot 10 mm.; taken in good artificial light.

He tells me he cannot see below, and demonstrates this loss with his hand in the lower part of the field quite intelligently; says he has to be constantly turning the eyes to the ground when walking. The fields exactly agree, and are typically those of altitudinal hemianopsia. He has right hemiplegia; considerable recovery has occurred, but the leg drags, and the arm remains very weak, with some blunting of sensation in the finger tips. He is totally unable to hear a watch with the right ear, but says he feels it ticking on contact; has had right facial paralysis, but it has now nearly entirely passed off. I take it the occipital lesion accounts for the visual defect, while the injury to the left Rolandic convolutions explains the hemiplegia, and possibly the facial weakness also. The left-sided damage might be in the posterior limb of the internal capsule as well as cortex. The deafness may be central, or more probably due to concussion of the internal ear, and it is possible that the right facial nerve may have been damaged in its passage through the petrous bone. Mr. Makins found all cases of bullet wound traversing the mastoid remained absolutely deaf, which he attributed to concussion of internal ear. In this case he considers the track through the brain no doubt involved a considerable extent of the outer aspect of the right occipital lobe, the cuneate lobule, possibly also the hinder part of the right temporo-sphenoidal lobe. It must have crossed the great longitudinal fissure and penetrated the left Rolandic region just above its centre. Mr. Makins considers the bullet entered at the occiput; the patient prefers to regard the more anterior wound as the aperture of entrance. There is no aphasia; the fundi, pupils, and movements of eyes all normal.

(*January* 31*st*, 1901.)

8. *A case of pulsating exophthalmos, following injury to the head.*

By D. J. Wood, Cape Town.

(Communicated by E. Nettleship.)

On May 26th, 1900, J. R—, a Johannesburg mine manager, came to consult me with reference to his eyes.

He gave the following history:—"A month ago, while cycling in South Wales, he was thrown from his machine. He fell on his head and became unconscious. When he came to himself he learned that he had lost a good deal of blood from his nose and forehead, and had been unconscious about twelve hours."

He felt better in a couple of days, and after seeing two doctors in London he left for South Africa, ten days after the accident.

When five days out—near Madeira—he began to feel a vague pain behind the eyes, and this quickly increased, so that for two nights he was unable to sleep. The pain was localised at the back of the eyes, and resembled a stretching of something in the orbits.

There had been no sudden feeling of anything giving way, and a bruit which came on at the same time did so gradually. He described the sound as like the exhaust of an engine; it was loudest in his left ear, and was synchronous with his heart-beats.

Coincidently with the orbital pain he observed that the eyes became prominent, and the right upper eyelid drooped gradually. He had double vision of the edges of everything; horizontal lines sloped down and to the left, and vertical lines fell over towards the left. During the same period his sight failed, especially in the left eye.

His state on May 26th was as follows:—There was a puckered scar on his left forehead, the right eyelid drooped, and there was great œdema of the eyelids. On looking at the

eyes one was at once struck with the engorgement of the conjunctival veins, which gave to the sclerotic a curious blue look, which I cannot recall seeing in any other case. The veins were not specially tortuous, but were large and numerous, a host of small vessels usually unseen being very easily discerned.

The proptosis being double was not very striking until one compared the eyes with a photograph, which showed them formerly to be rather deeply sunk under heavy brows.

The condition at once suggested a vascular lesion, which was confirmed by the forcible forward pulsation which one felt on touching the eyes.

The impulse was greater in the left eye, but even in the right was strong. There was no thrill. The stethoscope revealed a loud blowing bruit over the whole head, focussed, however, in the left orbit. The retinal veins were distended and dark; no hæmorrhages. One could trace with abnormal ease the small veins arising near the yellow spot. The optic discs were normal.

There was considerable though incomplete ptosis on the right side, less on the left, and also double, nearly complete, ophthalmoplegia externa, the right superior oblique, however, being exempt. In the photograph from the right side the eyes are opened *ad maximum*, as may be seen from the wrinkled brow.* Fields of vision full. Vision: right $\frac{6}{9}$, 3 letters; left $\frac{6}{36}$.

The diagnosis presented no great difficulty, *viz.* an aneurism by communication between the internal carotid and the cavernous sinus on the left side, causing arterial blood to be pumped into the orbital veins on both sides.

As it seemed likely to become a surgical case, I took the patient to see Dr. A. E. Thomson, who broadly agreed with me. After some consultation, the treatment agreed on was rest in bed, spare diet, and iodide of potassium, gr. v, thrice daily.

During the first week of treatment the eye symptoms

* Photographs were exhibited.

became worse, owing to very considerable chemosis and œdema of the ocular conjunctiva; the eyes could not be closed, especially on the right side, and the corneæ seemed in danger.

The chemosis, however, did not advance, and in the left eye, at least, began to improve.

During the time that patient was kept in bed his vision apparently grew worse in the left eye, and the field of vision was restricted; but as no complaint was made of this, and patient usually said he was "a little better," the actual condition was not noted.

Towards the end of July (after nearly two months' treatment) no definite progress had been made, and it was decided to operate. His vision in the left eye was now eccentric from the presence of an ill-defined central scotoma. The optic disc looked distinctly pale. His general condition was somewhat low, pulse small and rapid.

On the 3rd of August Dr. A. E. Thomson exposed and ligatured the left common carotid. This immediately stopped the pulsation in both orbits.

Manual compression of the carotid had been tried on several occasions, but on account of the muscular development of the cervical muscles was probably not enough to stop the blood-stream, and the pulsation seemed only lessened.

After operation the patient's mental condition was a little peculiar, his speech was thick and slurred, he was very irritable and easily excited. The result was, however, excellent. The next day the patient had regained power over the right upper lid, over the left internal rectus, and there was up-and-down movement in both eyes. The congestion of the conjunctival veins was gone on the right side and less in the left.

Owing to general restlessness the wound (after almost healing) broke down, and it was nearly a month before the patient could be out of bed. His normal mental state returned during the first week after operation, and vision in the right eye improved. In the left eye the central

scotoma diminished. The movements of the eyes became normal except for paresis of the left sixth nerve.

On the 17th of September (six weeks after operation) Mr. R— left for Johannesburg to resume his work. His condition was then as follows:—A very faint bruit audible sometimes at night; no pulsation; no proptosis. Both eyes open well, and the only muscular weakness is of the left external rectus causing a convergent squint. Vision: R. $\frac{6}{6}$; L. $\frac{3}{60}$. Right field of vision normal; left somewhat diminished with a central scotoma of about 15° radius, whose limits were very indefinite. The conjunctival veins were still dark. The ophthalmoscope showed the optic disc to be very pale and probably somewhat atrophic.

The only remarks I should like to add to the foregoing description are that it was a matter for regret that operation was delayed so long. It was to some extent due to the fact that the patient always said he was improving while in bed, and to the fact that neither Dr. Thomson nor myself were able to obtain any statistics of other cases to guide us in treatment.

One hopes that in similar cases as soon as the diagnosis is certain, the surgeon will proceed to operation.

(*May 2nd*, 1901.)

P.S.—After Mr. J. R— returned to work he tells me he had a good deal of pain over the left side of the head, especially when in bed at night. He thought it was due to the increased blood-pressure in unaccustomed channels. It gradually diminished and has not been felt for two months. The left eye remains somewhat prominent and congested on the surface, but Mr. R— thinks both conditions are improving. Its vision enables him to guide himself and to read the largest print at close range.

He states with much satisfaction that his right eye is in every respect as good as before the injury, and he can do as hard a day's work as he ever could.

(*July 27th*, 1901.)

The PRESIDENT said the chief matter for regret was that the operation, which was ultimately so successful, was postponed so long. When clinical assistant at Moorfields he saw a case of Mr. Hulke's (related in the Hospital *Reports*) which was absolutely cured by that means.

Mr. TREACHER COLLINS had a case of pulsating exophthalmos under his care two years ago. The patient was a man aged thirty-four years, who had been shot below his left ear in 1885. By means of the X-rays a bullet was located as situated near the base of the basilar process. There was a lobulated pulsating mass at the upper and outer part of the left orbit, and the left eye was much proptosed. He was much troubled by a buzzing noise in his head, which was so loud that he said his wife heard it at night. Pressure on the common carotid, which arrested pulsation, was kept up for several hours but without any lasting improvement. He was then transferred to Mr. Openshaw at the London Hospital, who ligatured the left common carotid. The patient did very well for a time, pulsation and the noise ceased; but when he (Mr. Collins) last saw him, about eight months after the operation, a considerable amount of the pulsation and the proptosis had returned. The noise, however, was not nearly so troublesome as before.

Mr. NETTLESHIP said he recollected a case of Mr. Hulke's about the year 1882, which was not permanently cured by that operation.

9. *Two cases of eye injury due to lightning.*

By M. T. YARR, R.A.M.C. (South Africa).

CASE I.—On one of my periodical visits to Cape Town in August last I was asked by my friend Mr. Granville, of No. 1 General Hospital, to see Major H—, a patient of his,

who was about to be invalided home for defective vision of the right eye. On examination I found right vision = fingers at three feet. Several large hæmorrhages—evidently not of recent date—and patches of atrophy in the retina in the region between the superior and inferior temporal veins, with a few scattered and much smaller hæmorrhages in the periphery, some engorgement of the veins, pallor of the disc, and contraction of the arteries. Left vision emmetropic; no lesions.

Major H— gave me the following history. He had been "trekking" with his column for the preceding ten months, and with the exception of an attack of jaundice in January lasting some three weeks, had enjoyed good health. In March, when camped in the Orange River Colony, a fearful storm burst suddenly over the encampment with remarkably vivid lightning; a flash struck his tent-pole, and at the same moment he felt something tingling down the right side of his head and down the right arm; he became half unconscious for a few minutes, but speedily recovered, although he now recollects he felt some tingling in the right side of his face and right arm for some days. His general health was good at the time. Four months later he discovered quite accidentally that he was practically blind in the right eye; he immediately consulted a medical officer at Kimberley, who invalided him to the base. There was no evidence of heart or kidney mischief.

On the face of it the evidence of cause and effect is by no means convincing, but read in connection with the following case, which my colleague on the train, Mr. Basil Pares, asked me to see, I think there is at least a strong presumption that the lesion in Major H—'s case was due to lightning stroke.

Case 2.—Mr. Pares has kindly furnished me with the following notes of this interesting case:—Richard Cox, æt. 25 years, a private in the 2nd Royal Berks Regiment, was employed on telephonic duties at Belfast, Transvaal. On October 18th he was sitting at an "operating table" at his

work, when, during a thunderstorm, the instrument which he was using at the time was struck by lightning. He experienced a 'shock,' and states that he fell back; on regaining his senses he was unable to see; no damage was done to his body or limbs; the instrument he was using at the time was, however, bent. The next day he says he could see light from dark, but nothing more. A doctor examined him two days afterwards, and he was admitted into a field hospital the fourth day after his injury; two days later he was transferred to Middleberg, and thence to Pretoria, where he became a patient of mine at No. 3 Model School Hospital.

On admission he had perception of light in both eyes, and could count fingers with the left eye. Tension was normal. On ophthalmoscopic examination, he was found to have a large detachment in his right eye, the detachment running in wavy whitish lines inferiorly.

Previous to his accident his vision in this eye was good; he was a second-class shot (there are three classes), and, as he puts it, "it doesn't pay to be a first-class shot in the army, as not only is more expected from you, but you are always pitted against the best shots in competitions;" but he tells me he scored a possible 28 at 800 yards at his last shooting course. I mention this as proof of his good vision in this eye.

His left eye also shows a large detachment, but spread over a larger area; his vision is, however, better in this eye.

There is so complete a history that there seems little room for doubt that the lightning was the cause of his loss of vision. (*June 13th*, 1901.)

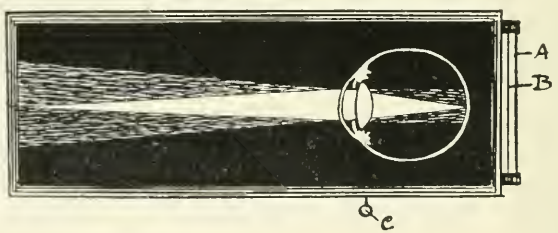
X. OPERATIONS, INSTRUMENTS, AND APPLIANCES.

1. *A diagrammatic model intended to assist in the teaching of ocular refraction.*

By H. Secker Walker.

The model consists of an oblong frame, thirty-six inches by fifteen inches, the background of which is painted white. In front of this is fixed a skeleton framework of a section of the eye about ten inches in diameter. By means of thin black slips of wood rotating on pivots behind the cornea, and worked in unison by a cord (A), the rays of

Fig. 8.



light (represented by the white background) emerging from the eye are made divergent, parallel, or convergent; while by similar means (B) the rays within the eye are focussed in the vitreous, on the retina, or beyond. The posterior half of the eye moves backwards and forwards to show the various states of refraction. To illustrate accommodation, the anterior surface of the lens can by a sliding rod (C) be made more or less convex.

The model can thus be adapted to show the course of the emergent rays in E., H., or M., and correcting lenses can be placed in front of the cornea to illustrate their action on light, and the reverse position of rays from distant or near objects can also be shown.

The model is intended for teaching purposes, and can be used for practice by students, so that they may get accustomed to carry in their minds the position of the rays of light in the different forms of refraction.

(*Card specimen. March 14th*, 1901.)

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2. *A capsulotomy knife.*

By Gustavus Hartridge.

This knife is made slightly curved, and has a sharp double-edged cutting point; the non-cutting portion of the knife is round, so as to fit the corneal wound and prevent

Fig. 9.

the escape of aqueous. The knife is intended to be entered at the corneo-scleral junction on the flat, and passed on till the round front of the shank is in the wound; the blade is then turned with the point of the knife towards the capsule or iris, which it will be found to enter easily, so that the necessary incision can be made.

(*Card specimen. March 14th*, 1901.)

In reply to the President, Mr. Hartridge said he had tried it in cases where the iris was drawn up into the wound. The knife never pushed the iris back. He cut

from above downwards, so as to get a vertical slit, entering it generally from below.

The PRESIDENT said he at one time used the vertical incision, but gave it up for the horizontal, because in the latter case the lower flap fell down and left a good opening.

3. *Recording colour perimeter.*

By J. H. TOMLINSON.

THERE are three special features :

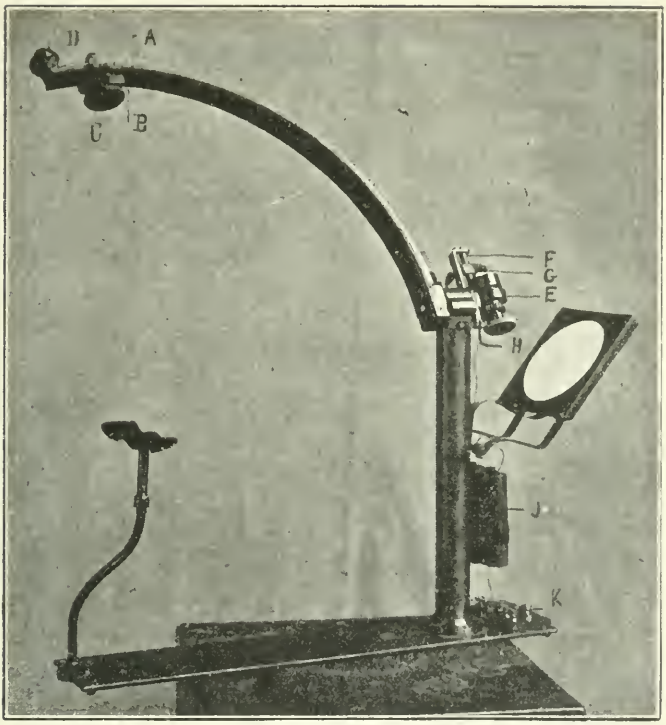
1. *Use of coloured light in testing.*—An electric lamp is arranged to slide along the quadrant. Caps containing coloured glasses or solutions can be fitted to the lamp as required. The intensity of the light can be varied.

2. *Adjustment by the patient.*—The slider carrying the lamp is operated by a cord attached by its ends to two drums, one at the end of the quadrant, and the other behind the axle of the quadrant, under the control of the surgeon. The first-named drum has milled edges, so that it can be turned by the patient to adjust the position of the lamp.

3. *Registering mechanism.*—The spindle of the back drum is geared to a rapid-pitched screw, on which works a nut carrying a pricking-pin. The diameter of the drum and the pitch of the screw are so proportioned that the pricker moves at one tenth of the speed of the periphery of the drum, that is at one tenth of the speed of the lamp carrier. By the simple expedient of transposing the gear wheels between the drum and the screw the relative speed is increased to one to six.

(*Card specimen. March 14th*, 1901.)

Fig. 10.



A. Slider carrying a box B containing a small electric lamp. C. Diaphragm with apertures ranging from 1 to 8 mm. D, E. Drums round which is wound a catgut cord connected to the slider and passing through the hollow axle of the quadrant. F. Screw driven by gearing from the spindle of the drum E. G. Nut riding on the screw and carrying a pricking-pin.

The current passes from the accumulator J to two springs H, bearing on insulated rings which are connected to insulated strips on the back of the quadrant. The terminals of the lamp bear by platinum-tipped springs on the strips. K is a switch board, with resistances to vary the power of the lamp.

The diaphragm C can be replaced by a series of diaphragms showing ordinary pigment colours.

(Made by Mr. Knoeferl, 26, Gosfield Street, Langham Street, W.)

4. *On peritomy in episcleritis, iritis, herpes ophthalmicus, and purulent ophthalmia.*

By T. Pridgin Teale, F.R.S. (Leeds).

Whether rightly or wrongly, I am under the impression that the value of peritomy, as an effective means of combating some of the more obstinate and baffling affections of the eye, has not been adequately appreciated in ophthalmic practice.

Under the name of "Syndectomy," or "Tonsure conjunctivale," it was introduced into practice by Dr. Furnari, of Paris, for the treatment of panniform and chronic lesions of the cornea, and for vascular ulcerations and interlamellar opacities (*Gazette médicale*, 1862, Nos. 4, 6, 8, 10, 12, and 14). The first notice in English literature, as far as I can make out, is an article on "Syndectomy," by Dr. Bader, in the *Ophthalmic Hospital Reports* (vol. iv, p. 19) for 1863, in which the author gives copious extracts from the original papers. Syndectomy was, as its name implies, an excision of a strip of conjunctiva and subconjunctival tissue, so as to denude and expose a ring of sclera around the cornea about one third of an inch in width.

It was discovered that this sacrifice of conjunctiva was unnecessary, and might at times be an actual disadvantage, and "peritomy" came in as a substitute. By "peritomy" I mean the laying bare by scissors of about one quarter of an inch in breadth of the sclera immediately surrounding the cornea, and the dividing all the vessels in that area which enter or emerge from the sclera, without any cutting away of tissue. This separated conjunctiva in some cases reunites so rapidly to the denuded surface and corneal margin that in severe cases I think it well to make two, three, or four radial cuts into the freed edge of conjunc-

tiva, so as to retard the reunion with the corneal periphery, and secure a more distinct cicatricial union as a means of cutting off and constricting vascular congestion.

The late Mr. Hewetson called our attention in Leeds to the remarkable effect of syndectomy or peritomy on the diffuse keratitis of inherited syphilis. In such cases I have employed it frequently, and I consider it to be the most powerful weapon we possess for combating this disease.

In this paper I wish to produce evidence of its value in episcleritis, iritis, herpes ophthalmicus, and purulent ophthalmia, and to raise the question of its possible use in rare cases for relief of tension.

I. Episcleritis.

The proved good effect of peritomy in diffuse keratitis naturally led me to put it to the test in episcleritis. This had been in my experience one of the most tedious, painful, and trying affections of the eye. Now with the aid of peritomy I undertake its treatment with much greater confidence in its early relief and complete recovery.

Let me give one or two examples.

Case 1.—Miss F—, a patient of Mr. Rowe, of Leeds. After long and patient treatment of a persistently recurrent episcleritis with no permanent effect, the right eye was peritomised on March 26th, 1897.

June 18th.—Now quite well. Has had only one very slight relapse.

February 24th, 1901.—Mr. Rowe reports that "since the peritomy there has been no serious relapse. The attacks became less frequent, milder, and have died out."

Case 2.—Miss G—. A very painful scleral patch near the upper margin of the cornea, with infiltration of the upper third of the cornea.

July 15th, 1898.—Peritomy.

July 27th.—All pain has ceased.

December 20th.—The corneal opacity is less by two thirds.

Case 3.—The following case is remarkable apart from the peritomy:—March 21st, 1900.—Mr. B—, a patient of Dr. Stewart, of Atherton. This was the most severe case I ever met with, the pain, however, being part of a general neuralgic disturbance of head, neck, and abdominal viscera. The neuralgia was "supposed to be a sequel of typhoid fever, which he got from rotten wool." The "awful" pain began in the eye and spread to the head, and later to other parts of the body. There was a red thickened patch of sclera rather less in size than a threepenny piece near the cornea. The conjunctiva was here so adherent to the sclera that it could not be dissected off, but had itself to be peritomised, leaving an island of adherent conjunctiva. The amalgamation, indeed, was so close that during the peritomy the sclera was opened by the scissors, and had to be stitched up. No harm resulted from the scleral wound, and the eye, in the words of his wife, "mended right away. As his eye got better the pain left his head." But pain developed about the diaphragm and stomach, and he died about six months after the operation.

II. Iritis.

Of the first case in which I did a peritomy for iritis I have no record. My recollection of the case is that after treating it for a considerable time, and obtaining full dilatation of the pupil, there was persistent pain with intense conjunctival and subconjunctival congestion. Being disappointed at the absence of improvement, I ventured to peritomise, knowing that it could do no harm, and hoping that it would do good. The result surpassed my expectations, as the pain forthwith subsided and the eye rapidly recovered.

Since that time I have several times used peritomy in iritis. The following case is a good example:—In April, 1895, a medical friend, in examining a female hospital patient, inoculated his finger with syphilis. Early in September, when away at the sea-side, the right eye became irritable, and on his return on the 11th iritis had fairly set in. The eye was at once put under treatment. On the 15th the artificial leech was applied to the temples. On the 18th he was put under ether, peritomised, and the artificial leech was repeated. On the 30th he resumed his work. February 26th, 1901.—My friend tells me, "Almost from the moment of the operation the severe pain ceased, and I lost the nocturnal pain and disturbance, which had been most distressing. I have never had the least trouble or sensitiveness of the eye since. It was a typical case of relief of symptoms."

A second case is worth recording, as I am convinced that the peritomy was the saving factor in the eye which recovered. Mr. B—, a patient of Dr. Robinson, of Rotherham, had been under treatment for double subacute iritis for about six weeks. There were synechiæ in both eyes. The right eye barely counted fingers, the left hardly distinguished shadows. May 29th, 1899.—Peritomy and iridectomy of both eyes. June 17th.—Right eye saw second hand of watch; left barely counted fingers. The right eye continued to improve; the left degenerated, and had to be extirpated. January 30th, 1900.—Right eye with optical correction read $\frac{20}{30}$ well; J. 4 well, J. 1 fairly.

This is the only instance, as far as I remember, in which I have combined iridectomy and peritomy, and also the only one in which I have ventured to iridectomise in the early subacute stage of iritis. The urgency of the case is shown by the fact that in the worst eye the disease went on unchecked by the double operation. Had I not felt that in peritomy there was an additional safeguard, I should not in such a case have ventured upon iridectomy.

III. Herpes Frontalis involving the Cornea and Conjunctiva.

There are probably few affections of the eye which are more unmanageable and more to be dreaded than the ulceration of cornea and congestion of conjunctiva which are associated with herpes. Since I came to appreciate the power of peritomy over congestion of the corneal periphery, two such cases have come under my care and have been submitted to the operation with encouraging results.

Case 1.—Mrs. G—, a patient of Dr. Gibson, of Boston Spa, had gone through an attack of herpes frontalis, which as it subsided left an exceedingly painful ulcer of the cornea with great conjunctival injection. June 16th, 1897. —Peritomy. After the operation the pain rapidly subsided, and the ulcer gradually healed.

Case 2.—Sister W—, æt. 69 years, a patient of Dr. Williams, of Richmond, Yorkshire, had suffered for six weeks from herpes of the nasal and supra-orbital nerves accompanied by paralysis of the external rectus. Two weeks before my visit, irritation of the eye set in with iritis and paroxysms of intense pain with quiescent intervals. Four days previously extensive marginal ulceration of the cornea appeared with aggravation of the pain. January 12, 1899. —Peritomy. 22nd.—Dr. Williams reports: "Much less pain since the operation. The ulcer has in a great measure healed, and the iris is clearing."

IV. Purulent Ophthalmia.

I have a case of purulent ophthalmia in which I am convinced that the eye was saved by peritomy.

On December 11th, 1899, I was asked by Mr. Snell, of Gargrave, to see the child of Mr. H— with this history:— It was born November 20th. November 24th the left eye

was inflamed, but under treatment the disease ran a moderate course and subsided. December 3rd.—Inflammation set in in the right eye, and in spite of treatment progressed as a subacute purulent ophthalmia.

After watching the case for four or five days the child was brought over to Leeds to be at hand for more active treatment should it be needed. The eye became steadily worse. The pupil, which at my first visit on December 11th was clearly visible, was now completely obscured by the opaque cornea, and during the preceding twenty-four hours a deep marginal ulceration had set in, and had spread almost to a semicircle in the lower half of the cornea. December 17th, at 9 p.m., I performed a peritomy. The degenerating process was at once arrested, and within a week the eye was recovering so distinctly that the child returned home.

January 5th, 1900, Mr. Snell wrote, "H—'s child is a wonder. For three or four days the cornea was very opaque, and then rapid improvement set in, and the dulness subsided. To-day the eye is nearly as bright as the other." July 3rd I saw the child, and found it difficult to detect which had been the affected eye.

V. Increased Tension.

After dealing with this question in the original draft of my paper, I had decided to omit the paragraph, as raising a doubtful point on very slight evidence. Since reading, however, in Dr. Bader's paper that Dr. Furnari had himself suggested "that syndectomy may replace iridectomy in some cases of glaucoma," it seems right to record the experience which I have had. The case was as follows: Mrs. S—, a patient of Dr. E. Colby, of Malton, had lost the right eye from irido-cyclitis accompanied by increased tension, which an iridectomy by a skilled ophthalmic surgeon had failed to arrest. Subsequently the eye was excised. She had an insuperable dread of an iridec-

tomy in the other eye, which was beginning to suffer in like manner, with T. + 2, vision reduced to J. 14, and a partially active pupil. September 26th, 1900.—Peritomy. October 5th.—There has been steady improvement. Reading J. 8, + T. ½. November 26th.—Vision has fallen off. Tension normal. Later still vision became reduced to perception of large objects.

In this case peritomy checked for a time the disorganising process and reduced tension, but did not permanently arrest the mischief. (*March 14th*, 1901.)

The PRESIDENT said possibly Mr. Teale had not seen a paper by his (Mr. Critchett's) father on peritomy, in which he gave some remarkable results which he could himself verify. The operation they both did, which he had called alternately "syndectomy" and "peritomy," consisted of removing tissue, but certainly not so much as one third of an inch; one sixth of an inch would be nearer the truth. He cut down to the sclerotic, and he attributed the good result to the cicatricial scarring, which virtually placed a species of pathological Esmarch's bandage over the vessels which were encroaching on the cornea. The operation was almost confined to complete or partial pannus and vascular ulcer, in which cases he had obtained very remarkable results, and had seen no bad effect. After the paper they had just heard, no doubt many of them would go on with the operation. He did not advocate the maxim, "When in doubt play peritomy," but the number of cases mentioned would encourage them to carry out the operation, especially for episcleritis. He said he could understand that in interstitial keratitis the scar would have but little effect, but in the cases where tissue was removed, if the operation were well done, the process was exceedingly interesting. About a fortnight afterwards the scar could be seen commencing, and at each subsequent fortnightly period the vessels could be seen strangled as they went across.

Mr. CARTWRIGHT thought with regard to the explanation

suggested by the President, namely, that the cicatrix acted as a sort of Esmarch's bandage, diminishing the blood-flow, that, however obvious it might seem in corneal cases, it was by no means satisfactory in cases of scleritis and episcleritis, where the affected area is outside the ring of scar tissue. It was still more difficult to explain by any such mechanical theory the benefit arising in cases of high tension. Might not the effect in these, if not in all, cases be due to the peritomy acting simply as a counter-irritant?

Mr. JOHNSON TAYLOR asked whether Mr. Teale ever did a partial peritomy. He mentioned one case of what was called sclero-keratitis, opacity of the cornea at one spot in continuity with the scleral patch. In such a case would Mr. Teale do a complete peritomy, or confine it to the affected locality? Last year he (Mr. Taylor) had a very troublesome case of recurrent trouble in the cornea of a healthy man. After trying all sorts of things, the patient saw an eminent London authority, who suggested cauterisation of the episcleral tissue, which he did, with very gratifying results.

Mr. HERN said that fifteen years ago Mr. Teale drew his attention to the operation, and during the last ten years he had done it eighty-six times. He usually cut away a piece of tissue about two lines from the margin of the cornea; then he took care that he got quite to the edge of the cornea. The results were not so good where he neglected that matter. With a sharp curette he curetted round the seat of operation, so that if any little vessels had been missed they might be torn across. His results from the operation in interstitial keratitis had been wonderfully successful, having stopped the process and prevented that intense opacity of the cornea which had resulted in most of the severe cases he had treated without surgical intervention. His episcleritis cases had numbered about thirty, and in every case there was benefit from the operation. He felt that it was a neglected operation at the present time.

Mr. SECKER WALKER said Mr. Teale had shown him the

value of peritomy fifteen years ago, and he had seen him do it many times. At that time he believed it was chiefly used for interstitial keratitis and corneal ulceration. Since then he had regularly performed the operation for keratitis, deep phlyctenular corneal ulcers, and episcleritis, and considered it of great value. He thought perhaps its least use was in interstitial keratitis (the deep form). For this disease he had often treated both eyes, one with peritomy, and one by the ordinary mercury and atropine treatment, but had never been able to decide in which cases the operative treatment was going to be of value. In some it seemed to do much good, in others little or none at all. Mr. Teale had mentioned that the success of the operation lay in formation of a pericorneal cicatrix. Mr. Walker did not quite agree with this because in most cases of peritomy a few months afterwards no cicatrix was visible. He believed it frequently acted as the very powerful and prolonged application of a leech would do. He had tried it lately on Mr. Teale's suggestion in three cases of iritis without marked success; one of these, however, was an old case of recurrent iritis, from which a good result was hardly to be expected.

Mr. Cropper said he had used peritomy in a case of obstinate pannus in a man of sixty, causing very defective sight, though with but little result. Though no "hairs" could be seen, the man begged hard for the operation for trichiasis, which was done (modified Snellen) on both eyes, with a most gratifying result, the cornea six months later being quite clear. He felt it was most important to treat the cause of pannus.

Mr. Teale, in reply, said the great point was to remove no tissue. If the case was severe, in order to prevent the too rapid reunion to the margin of the cornea, he made the vertical cuts whereby the elasticity of the conjunctiva drew its cut edge farther away, and retarded healing. He had seen harm in one or two cases where there was extensive ulceration of cornea in a rapidly degenerating eye, where the peritomised conjunctiva came over the cornea

and seemed to grow to the ulcerating surface, and the whole went on from bad to worse. That happened in both eyes of one man. With these exceptions he had seen no harm result, and inspection, unless very close, a month or two afterwards would hardly detect any trace of the operation.

In reply to Mr. Johnson Taylor, where the congested area consisted of one fourth of the circumference, he did a partial peritomy. Where there was any doubt he believed one should go all the way round. With regard to using the small curette for scraping the denuded surface, he had done it on many occasions and thought it beneficial, especially where the part was intensely congested. Sometimes, where peritomy was done for long-continued ulcer of the cornea, the conjunctiva was almost adherent all the way round, it was not ordinary loose conjunctiva. Whatever the explanation of the remarkable effect of the operation, there were the facts that vessels were depleted and cut off, that there was more or less cicatricial reunion, and a definite disturbance of the nature of counter-irritation. The operation was an addition to the surgeon's armamentarium, which, though it did not succeed in all cases, was sufficiently effective to take a great place in treatment. He was not aware of the contribution on peritomy by the President's father, and he would be very glad to hear of any further papers on the subject, as there might be others which he had overlooked.

5. *A portable Refractometer.*

By C. Blair.

The instrument consists of a frame which is placed on the patient's face like an ordinary trial frame, but is reversible, and carries on each side, *i. e.* before each eye,

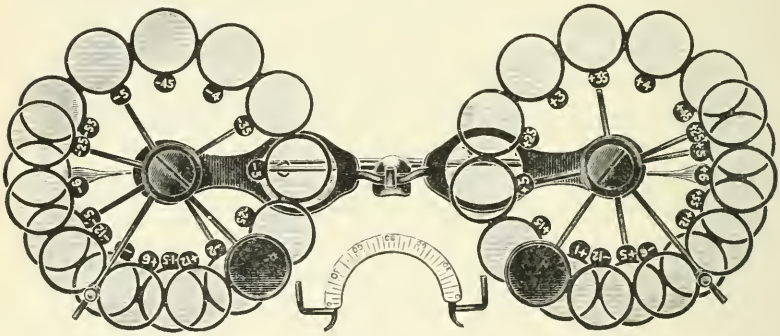
revolving lenses mounted on a primary and secondary wheel (or sector of a wheel).

On one side are mainly *plus* spheres, those on the primary wheel ranging by half-dioptres up to + 5·5 D., and on the secondary wheel + ·25 D., + 6 D., + 12 D., − 6 D., and − 12 D.

On the other side are mainly *minus* spheres, those on the primary wheel ranging by half-dioptres to — 5·5 D., and on the secondary wheel — ·25 D., — 6 D., — 12 D., + 6 D., and + 12 D.

By this arrangement it is possible to place before the eye *plus* or *minus* spheres, ranging by quarter dioptres up to 6 D., and above this by half-dioptres up to 17·5 D.

Fig. 11.



(*i.e.* ninety-four different corrections). Moreover by means of the + 6 D. and + 12 D. lenses on the *minus* side, and the − 6 D. and − 12 D. on the *plus* side, it is possible by superimposing to have *plus* spheres before both eyes simultaneously, or *minus* spheres before both eyes, and so get binocular correction.

The instrument is chiefly intended to save time and trouble in retinoscopy, but may also be used for subjective testing in all cases of simple hypermetropia, myopia, and presbyopia.

Messrs. Curry and Paxton have kindly made the refractometer for me. It was intended to have an arrangement

for holding ordinary cylindrical trial lenses, to be rotated in a graduated cell in the usual way. There was a difficulty in fixing this on the present frame, but it will be on the next instrument, together with some other modifications. Each wheel is five inches in diameter, and the whole weight of the instrument is eight ounces; but the next, which is to be made of aluminium, will be lighter.

(*Card specimen. May 2nd*, 1901.)

Mr. G. HARTRIDGE suggested that space would be economised by having lenses half the size of the present ones.

The PRESIDENT said he thought the idea might result in something useful by-and-by. He regarded Mr. Hartridge's suggestion as a good one.

6. *Thiersch grafting for xerophthalmia.*

By ARNOLD LAWSON.

THE patient came to the Royal London Ophthalmic Hospital at the commencement of 1900, suffering with a chronic inflammatory condition of both conjunctival sacs, associated with considerable shrinking. He stated that his eyes first became inflamed in October, 1899. The shrinking of the conjunctiva progressed very rapidly in spite of lotions and ointments, and by March, 1900, there was almost complete symblepharon between the lower lid and the globes, and the cornea was threatened in each eye.

The operation on the left eye was performed in April, and that on the right eye in May, 1900. In both eyes the same method was adopted. It consisted in dissecting the shrunken conjunctiva off the lower lid, and placing a large Thiersch graft cut from the arm over the raw surface of the lid. The graft was kept in position

in a way suggested by Mr. Stanford Morton, *viz.* by four sutures arranged in a criss-cross fashion in pairs, so that the length of each suture lay across the graft, whilst each extremity perforated the graft and thickness of the lid, and was tied over a piece of rubber tubing.

The grafting has had the effect of completely alleviating the symptoms, although shrinking is still going on, as can be seen in the way the grafts have been drawn over the corneal margin. (*Card specimen. June 13th*, 1901.)

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7. *An apparatus for the performance of vibration massage, as adapted for the treatment of eye diseases.**

By Ainslie Hudson.

The instrument is driven by an electric motor, which is worked by the constant current of 100 volts, by the same method that a reading lamp would be worked from the main. The motor causes the shaft inside the flexible tube to revolve, on the same principle that a dental engine would be driven. At the end of the flexible tube is an air-tight metal box, in which revolves an eccentric block, the revolutions of which set up rapid vibrations, which are transmitted to whatever end-piece it is desired to use. These end-pieces, some of vulcanite and some of rubber, are applied to any surface that it is required to massage, whence the vibrations are transmitted to the subjacent parts. By different adjustments of the handle and the metal arm into which the end-pads are inserted, any kind of vibration can be obtained, from the faintest stroking to the coarsest hammering. That these vibrations penetrate to a great depth I have demonstrated by experiments on different

* Invented by Buckhardt, of Leipzig, and imported by Isenthal and Co., of Mortimer Street, London, W.

columns of water enclosed in india-rubber bags, even moderate vibrations penetrating practically through the body. Moreover, they can be transmitted to parts quite inaccessible by the ordinary methods. The instrument is, I believe, comparatively little known in this country. I have seen only one other, that had been brought over from America, but it was of a much more primitive construction than the one I show.

Vibration massage is merely a new adaptation of an old idea—the adaptation of electricity to bring about mechanical movements as a substitute for the same movements produced manually.

As to the physiological effect of these vibrations, I think that there can be no doubt that molecular changes are produced in muscular and nervous tissues, that the circulation is stimulated, whereby the nutrition is improved. Moreover, the lymphatic circulation is stimulated by the process, as I have had opportunities of noting in cases of acute and chronic inflammatory conditions. The same may be true of manual massage, but it is not every one who has the faculty or the time or the patience to practise manual massage, whereas anyone can apply this vibration massage, which, as far as the mechanical part goes, is as thorough, if not more so, as any that could be performed by hand, besides being applicable to parts inaccessible by the ordinary manual methods.

As to the cases in which I have employed it with benefit, and which, I think, may be taken as fair examples of the class of cases in which massage is indicated, I shall enumerate them immediately. I may say that many of them were having no additional treatment at all during the time that they were having the massage treatment, and that those who were under other treatment at the time seemed to make a fresh effort to get well soon after the vibration was commenced. In a few instances the patients had been under ordinary treatment for months without any apparent progress. In seven cases of glaucoma, acute and chronic, the tension was lowered in a few minutes, never less than

one degree, and sometimes from T. + 2 to normal. In one instance this was maintained for an hour without the employment of eserine, and in another for two hours. The other five I was unable to watch, as they came as out-patients and left soon after the treatment. Two cases of severe irido-cyclitis have had the pain relieved, and have quieted down in a few days, including two cases following cataract extraction, in both of which instances the eye was regarded as lost. In a case of interstitial keratitis of long standing the corneæ have undoubtedly cleared after a fortnight's treatment; and although the vision was not tested at the beginning of the treatment, the patient says that he can see much more clearly. Other treatment is in abeyance.

A case of incessant supra-orbital neuralgia and insomnia lasting for nine weeks had two applications. After the first she was relieved of the pain and had a good night's sleep, and after the second she was permanently relieved. I saw her a month later, and she had had no return of either symptom.

A patient with paralysis of the left internal rectus, with diplopia for two and a half years, after a fortnight's treatment by massage lost most of his diplopia, and could rotate his eye inwards about 25°, and this without any other treatment for nine months. On testing him with the finger I found that he had single vision even when looking at the object at the extreme limit in the direction of the paralysed muscle.

A case of paralysis of the external rectus is beginning to get occasional single vision, other treatment being in abeyance. In a case of nebulæ of the cornea where I have tried massage, the nebulæ have become less dense, and two cases of follicular conjunctivitis and strumous keratitis respectively that had remained without alteration for a long time improved rapidly under the massage. A case of optic atrophy had his fields increased to more than double after two sittings. In every instance pain has been relieved temporarily or permanently, and patients have often stated

that they have had their first night's good sleep after the massage.

It may be said that this treatment has not been sufficiently elaborated to warrant my demonstrating it at this early stage. My excuse is this, that before the next session of this Society commences, I shall have returned to my practice abroad, and this is probably the last time that I shall have the privilege of attending its meetings. I claim no supernatural powers for the instrument, but merely show it on its merits, in the hope that some may be induced to adopt it and give it a trial, for judging by some of the results that I have already obtained during a period of only a few weeks, I am confident that it will be found of value in the treatment of eye diseases. In conclusion, I may state that I have in each instance endeavoured to get an honest report from patients. I have put no leading questions, but merely asked what their impressions have been, and these I have noted down at the time.

(*Card specimen. June 13th*, 1901.)

8. *New pattern of eye speculum.*

Devised by Herbert E. Drake-Brockman, I.M.S., and exhibited by E. F. Drake-Brockman.

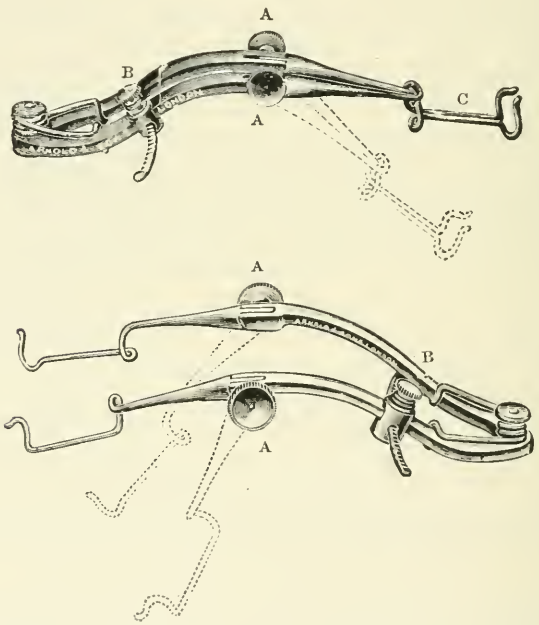
The chief advantages claimed for this instrument are the following:

First: That it is made of the thinnest tempered wire compatible with rigidity, thereby making the spring of the instrument more pliable, which latter, in consequence, is of light weight.

Second: That it allows of considerable alteration of its general curve to suit the contour of any human face, in which position it can be readily fixed by adjustment of the

screws (A A), the heads of which latter are cupped and of sufficient size to admit of a firm grip of the speculum by the fingers during manipulation, thereby allowing of free and unfettered use of instruments from the side of the face, as is necessary in the performance of the operation for extraction of cataract, etc. It will be noted on inspection that the screws A A have been made so that their threads are reversed, with the object that, by screwing

FIG. 12.



against each other, it is easy to adjust the speculum at any angle while within the eyelids, and fix it there, thereby not necessitating its withdrawal prior to adjustment. The body of the instrument can be made of aluminium if preferred, which will render it lighter. The prongs themselves which catch the eyelids, I fear, cannot be made of that metal, as it is incapable of being bent, but in my speculum

the thinnest wire compatible with rigidity has been employed for this purpose, and has answered very well indeed.

Third: That it allows of absolutely complete closure to the thickness of only one wire's breadth, by the telescoping of one prong into the other, thereby allowing of instant removal from the eyelids, by simply compressing the limbs at the buttons A A. This important feature in the instrument is its chief virtue, and one of the greatest value in the event of a nervous patient straining after the extraction of the lens in cataract operations, or when extrusion of the vitreous is feared, as the pressure can be instantaneously relaxed by adopting the above-mentioned manœuvre, with the result that the speculum of its own accord falls out at once from between the eyelids.

Fourth: The natural curve given to the instrument has been arrived at as the result of a number of experiments made by me on the living subject, and that depicted in the figures is the one, I find, which is adaptable to the faces of most ordinary adult individuals. This is, however, capable of further alteration, if necessary, to any angle required, by adjustment of the buttons A A, which are, in reality, screws, for fixing the limbs of the instrument at any angle, as well as for the purpose of admitting of a reliable grip of the speculum to prevent any chance of slipping during manipulation between the eyelids, in the performance of operations on the eye.

Fifth: The screw with rack (B) for regulating the extent of the opening of the speculum, has been placed as far back as possible at the lower extremity of this instrument, and made purposely of small dimensions (more so than is usually the case with the ordinary eye speculum) so that it can in no way interfere with the free passage of instruments from the side of the face.*

(*Card specimen. June 13th*, 1901.)

* The speculum is made by Messrs. Arnold and Sons, Smithfield, London, E.C.

The PRESIDENT said the members were indebted to any one who would give them an instrument that would fall instantly out of the eye when extrusion of the vitreous was threatened. He was inclined to ask whether, with the additional hinges, the apparatus could be kept aseptic. He thought it would be a valuable instrument, and he would be pleased to experiment with it.

XI. CONGENITAL CONDITIONS.

1. *Congenital malformation of eyelids, with symmetrical lacrymal fistulæ.*

By Harold B. Grimsdale.

V. F—, æt. 7 months. The child is suffering from great malformation of the lids of both eyes. Besides the small palpebral fissures, and the marked epicanthus, there is on each side a fistula at the lower part of the lacrymal sac. There has been at no time any discharge from the eyes. The little fistula on the right side was noticed soon after birth; the more minute left fistula was overlooked.

There was no ocular trouble until about one month ago, when the margin of the right fistula became a little irritated and eczematous.

The left fistula is extremely small and hardly any escape of fluid takes place from this side.

The small amount escaping by either fistula does not seem to account for the whole lacrymal secretion, and there is no accumulation in the sac, from which I infer that the duct is probably patent.

(*Card specimen. October 18th*, 1900.)

2. *A case of congenitally imperfect separation of iris from back of cornea; rise of tension induced by use of a mydriatic.*

By J. Herbert Fisher.

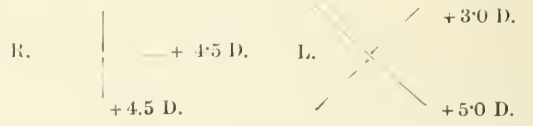
R. G—, Jewess, æt. 27 years. Came to me at Moorfields Hospital November 17th, 1900. The following history was elicited a week later. Three weeks ago she had been in the London Hospital, in the Jews' ward, on account of pain in the left eye. Before going into the hospital she had noticed coloured rainbows round artificial lights; drops were used at the London Hospital, and she took out drops "which became red on keeping."

She complained to me of pain in the left side of forehead.

On examining the left eye the whole anterior surface of the iris is rugged and irregular with excavations. Around the whole outer margin of the cornea there is a narrow crescent of pigmented iris tissue adherent to the back of the periphery of the cornea; delicate strands of iris tissue pass from the anterior surface of the iris on to this detached slip, the latter being obviously split off the anterior surface of the iris. On the inner side there is a similar but smaller slip of iris tissue adhering to the back of the cornea at its periphery, but this crescent of tissue is less wide than that on the outer side. There is no congenital pupillary membrane; the pupil acts normally but is slightly excentric. T. n.

The right iris is normal in every way.

On November 21st I used homatropine and cocaine drops three times to both eyes. She reads no English. The corrected retinoscopy gave—



I tested the T. both before and after the mydriatic, but detected no increase. Ordered R. and L. + 3·0 D. sph., constant; subjective tests of little value, but apparently she did not receive benefit in V. from a cyl. in L.

Possibly the astigmatism in the L. is evidence of faulty corneal development.

Three days later she returned. The night following the use of the mydriatic she got increased pain in the L. eye and forehead, and the eye became red. She saw no coloured haloes. When I saw her the R. pupil had contracted after the mydriatic, but the L. was unduly dilated still. There was injection of anterior ciliary veins of the L., and T. was + 1 (confirmed by Mr. Lawford and Mr. Holmes Spicer). I used eserine once; it caused much severe browache, but contracted the pupil readily, and the T. fell almost to normal in three quarters of an hour. The congested veins remained. I ordered pilocarpine drops (1 per cent.) three times a day to the left only.

December 1st, 1900.—L. T. n. L. P. = 5½ mm. R. P. = 4½ mm.

8th.—L. T. n. L. P. still ½ mm. greater than the R. P. Field full on perimeter. Fundi are both n. There is no cupping of L. O. D. or pulsation of vessels. The A. C. of L. is, and has been always, of full normal depth.

The case is of great interest as bearing on the origin of congenital glaucoma or buphthalmos. The features briefly are: Blocking of the approaches to the canal of Schlemm by a congenital imperfect separation of iris from back of cornea. Threatening glaucoma, which a mydriatic will induce in this eye, but not in the R., in which the iris is normal. Astigmatism in this eye only, perhaps indicative of the faulty development of cornea.

(*Card specimen. December 13th*, 1900.)

3. *A rare example of persistent capsulo-pupillary membrane.*

By G. Brooksbank James.

(With Plate VI, figs. 1 and 2.)

C. F—, æt. 8 years, female, attended the Royal Eye Hospital in November last, as parents wished advice for a squint, and complained that the child had defective vision in the right eye.

The family history is good. Patient is the second child in a family of four children, none of whom, on careful examination, disclose any abnormality with regard to the eyes or other parts of the body. There is no history of miscarriages, and parents have enjoyed good health. The patient herself was a full-term child, and the eyes seemed quite right until fifteen months of age, when the right eye began to squint. She had attended the Royal Eye Hospital two months previously to this for an attack of inflammation in the right eye. She only paid two visits to the hospital, at intervals of a week, and was practically well at the second visit. The mother has never noticed anything peculiar about either eye, with the exception of the squint in the right eye; she states, however, that up to three years ago the child could distinguish objects such as money, etc., quite well with the right eye while the other was covered, and that up to twelve months ago, when the left eye was occluded, the child looked straight at her with the right eye, but that lately she has been unable to do this.

Present condition.—R. V. $\frac{6}{nil}$ with perception and accurate projection of light. L. V. $\frac{6}{6}$ with + 5 D. sph.

The right eye shows a convergent strabismus of 40°. The cornea is slightly below the normal measurements, but exhibits no opacity. The iris is of a pale, greyish-yellow colour. The site of the normal pupil is occupied

PLATE VI.

Figs. 1 and 2 illustrate Mr. G. Brooksbank James's case of a Rare Example of Persistent Capsulo-pupillary Membrane (p. 168).

Figs. 3, 4, and 5 illustrate Mr. F. R. Cross's case of Congenital Fibro-vascular Tumour of Eyeball (p. 178).

M. Taylor, del.

Fig. 1.

Fig. 2.

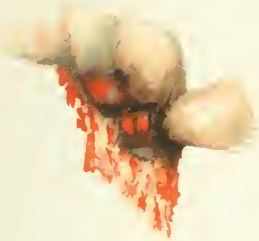


Fig. 5.

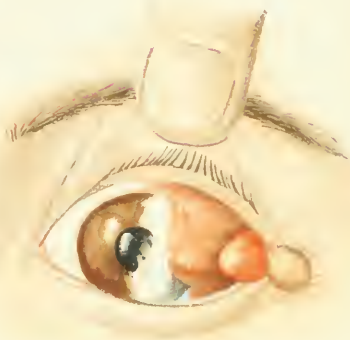


Fig. 3.

Fig. 4.

W. N. Blatchford, del.

Bale & Danielsson, Ltd., Chromo-Lith.

by some six or seven strands of tissue closely resembling the iris in structure, and arising apparently from the circulus minor (fig. 1). The strands converge to the centre of a whitish disc-like opacity which is situated behind them. They enclose some half dozen small oval openings the largest of which is situated slightly above and internal to the normal situation of the pupil. It is 1 mm. in length, and $\frac{3}{4}$ mm. in width when not under the influence of a mydriatic, but 2 mm. in length and $1\frac{1}{4}$ mm. in width when under its influence. The other small openings, all below 1 mm., react to atropine in a similar manner. They all react consensually to accommodation of the other eye for a near object. The left eye (fig. 2) presents a strand of pupillary membrane at the inner margin, and there are also two free tags present at the outer part.

There seems no reason to suppose that the abnormality is due to a pre-existing iritis, intra-uterine or otherwise. The fact that a remnant of the pupillary membrane is also present in the left eye inclines one to consider the whole condition as congenital. (*December 13th*, 1900.)

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4. *A large area of retinal pigmentation below the disc.*

By G. Brooksbank James.

(With Plate IV, fig. 2.)

F. M—, æt. 11 years, schoolboy, who attended the Royal Eye Hospital for defective visual acuity, was found, under atropine, to have V. = $\frac{6}{6}$ with + 6·5 D. sph. in each eye. Visual fields both good, there being a relative scotoma in the left eye corresponding to the patch described below.

There is a congenital crescent along the lower and inner margin of the left disc. Extending from this downwards and inwards is a large sector-shaped patch of retinal

pigmentation. The pigment is chiefly situated beneath the vessels, although close to the disc the latter seem to dip into it. It is very uniform close to the disc, but more discrete towards the fundus. The latter is normal elsewhere. There is a strand of pupillary membrane present in this eye. The case is apparently somewhat similar to others described by Mr. Sydney Stephenson in vol. xi of the *Transactions*. The pigment in these cases was, however, more discrete, though in two of them it occupied a sector-shaped area below the disc.

No very satisfactory explanation has been given as to the cause of such a condition. Mr. Frost (*Brit. Med. Journ.*, December 14th, 1899) states that such cases are probably analogous to the sectors of pigment met with in the iris. That, however, is not exactly an explanation of the condition. It is generally thought that the pigment patches in the iris are derived from cells of mesoblastic origin in its stroma, and there can be no analogy between them and retinal pigment derived from the optic cup. There is no reason to suppose that these cases are akin to pigmentary retinitis; the family and personal history, examination of fields, etc., negatives such a conclusion. The situation of the pigment in the present case tempts one to think that it may in some way be connected with the closure of the optic cup. (*December 13th*, 1900.)

Mr. Adams Frost said, with regard to Mr. James's suggestion as to the pigment being derived from the optic cup, in two cases, he (Mr. Frost) had seen the patch of pigment above the disc. He had not seen many such cases, and therefore did not know that there was a great preponderance of cases in the lower region. The arrangement of pigment was different from that of retinitis pigmentosa. There were no processes jutting out, the pigment spots were more or less quadrilateral, and were arranged in groups of five or six, and sharply defined.

5. *Coloboma of optic disc (left).*

By J. Herbert Parsons.

L. W—, æt. 10 years, has never seen well with left eye.

Vision.—Right eye $\frac{6}{6}$, J. 1, Hypermetropic + 3 horizontal + 2 vertical.

Left eye $\frac{6}{60}$, J. 15, Hypermetropic + 5·5 horizontal + 3·5 vertical; vision improved to $\frac{6}{24}$ with correction.

Right fundus normal; right field normal; normal tension.

Left fundus, disc much enlarged and cupped; tension normal.

Depth of cup = 2 D., although it gives the impression of being much deeper. All the vessels are at the edge of the disc, principally above and below, except two fine arteries arising from one of the upper vessels; fundus elsewhere normal.

Left field nearly as large as right.

Blind spot could not be accurately mapped out, owing to age of patient, but is larger than normal.

(*March 14th*, 1901.)

6. *A case of high hypermetropia.*

By H. Work Dodd.

A man æt. 38 years.

Retinoscopy under mydriatic gives—

+ 15 D.	+ 15 D.	+ 16 D.	+ 15 D.
R. E.		L. E.	

V. R. $\frac{2}{60}$ J. 10 at 10 cm.; L. $\frac{2}{60}$ J. 10 at 8·3 m.

After retinoscopy:—R. $\bar{c}$ + 15 D. = $\frac{6}{60}$, and J. 8 at 8 cm.; L. $\bar{c}$ + 15 D. sph. with 1 D. cy. axis 50° down-in = $\frac{6}{60}$ and J. 8 at 8 cm.

The lenses are present. There is a distinct lack of power of convergence.

His son, æt. 3 years, shows (under a mydriatic) hypermetropia + 14 D. in both eyes.

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7. *Dermoid of conjunctiva with fissure of lid.*

By William Lang.

At the outer canthus there is a small notch in the lower lid corresponding to a dermoid growth of the conjunctiva, which extends from the region of the lacrymal gland downwards and inwards to below the cornea on its inner side, covering the lower third of the cornea.

At the upper and outer part the growth consists of the usual fibro-fatty tissue with thickened white epithelial masses on the surface, but no hairs can be seen. On the cornea it is very thin and vascular, like scar tissue, but it is freely moveable and is adherent only towards the centre of that membrane.

At the upper and inner margin of the cornea, astride the limbus, are two small dermoid growths, about 2 mm. in diameter.

The eye converges. (*May 2nd*, 1900.)

Mr. Johnson Taylor asked whether Mr. Lang proposed doing anything in his case. He (Mr. Taylor) had a similar case of dermoid growth freely moving over the sclerotic, and he thought it would readily be got rid of. But when he got halfway through the operation vitreous escaped, so he removed only the most prominent portion and stitched up the rest.

8. *Coloboma of eyelid with deformity of auricle and dermoid growth.*

By W. Adams Frost.

A. H—, æt. 19 years.

In right upper lid towards the inner side a quadrilateral gap measuring 10 mm. horizontally and 6 mm. vertically. The eyelashes here are absent. Above the eyebrow, corresponding to the gap, is a single tuft of long hairs. The vision of the right eye is somewhat defective from uncorrected hypermetropic astigmatism; but the eye appears to be normal in all other respects.

The caruncle is displaced downwards, so that it appears to be connected with the lower lid only; protruding from beneath the outer part of upper lid, and covered by the conjunctiva is a firm, moveable growth. This appears to be similar to the swellings noticed in several other cases of coloboma of the eyelid (Juler, *Trans. Ophth. Soc.*, xii, 171; Snell, *ib.*, iv, 348; Cowell, *ib.*, xi, 214. In this last case the growth was excised and found to be dermoid in character.)

The right auricle is small and misshapen; there appears to be no external auditory meatus. The patient considers this ear to be quite deaf, but a watch applied to the head on that side is heard, apparently with the right ear. There is a so-called supplementary auricle on the left side. External to the right angle of the mouth is a small nodule; on the left in a corresponding position is a dimple. The patient thinks that something was removed here by operation.

The co-existence of deformities of the auricle, and the presence of dermoid tumours in the position of the mandibular clefts appear to be frequent in coloboma of the eyelid. Nicolin's *Mémoire* published in 1888, contains a description of fifty-six cases of congenital coloboma of the eyelids.

(*May 2nd*, 1901.)

9. *Two cases of congenital ptosis upon which a modified Hess's operation had been performed.*

By G. W. Roll.

(With two figures.)

The annexed notes on some cases in which I have performed the operation for ptosis designed by Hess are intended to bring into prominence a modification which appears to be of importance in regard to the effect attained. I show two patients to the Society this evening. In the elder the operation was performed three years ago on the left upper lid and eyebrow. I am able to show the photograph of this patient taken before the operation (Fig. 13), again a photo taken three months after operation (Fig. 14), and also the patient in person to-night.

The younger patient has had the operation performed on both right and left eyelids, and this occurred some twelve months ago, with a short interval between the two operations.*

The steps of the operation cannot be better described than by quoting Mr. Lawford's short account in the *Ophthalmic Review*, vol. xiii:

"The eyebrow having been shaved, a curvilinear incision is made in its whole length, and following its course, so that the scar may be hidden by the subsequent growth of hair. This incision is carried through the skin and subcutaneous tissues. Starting from this incision the skin of the upper eyelid is separated from the underlying layer of the orbicularis palpebrarum, the dissection being carried down

* I have to thank Mr. Henry Juler for placing at my disposal the elder of these two patients at the time when I was working as his clinical assistant, and for permission to present him to this meeting.

nearly to the ciliary margin and along the whole length of the lid. Three silk sutures, each one armed with two needles, are then required, one at the centre, and one towards each end of the lid and about halfway between the eyebrow and the lid margin. The needles are passed from without inwards, through the skin only, and are brought out in the space made by the previous dissection. The two needles of each thread are inserted about 5 mm. apart. When these stitches are pulled upon the skin of the upper lid becomes folded upon itself, the fold corresponding fairly accurately to that normally present in the lid. The needles are now passed deeply under the upper border of the incision in the brow and brought out a few millimetres above. The two ends of each thread are tied over a small roll of plaster (or, better, rubber tubing); the amount of traction necessary being gauged before the knots are made fast. The wound in the brow is united by a continuous suture. The stitches are left in from eight to ten days, and can, if necessary, be tightened from time to time."

But anyone who performs this operation according to the above directions will find that if the needles are inserted "about halfway between the eyebrow and the lid margin," he will not obtain enough effect, and that to get more the needles should penetrate the skin and leave the sutures much nearer the ciliary margin. When tightened the upper lid will no longer be in contact with the globe, but gives the impression of being slightly everted. The elasticity and stretching of the thin cutaneous covering of the upper tarsal plate rapidly compensates for any over-effect which may at first cause apprehension. In fact, to secure the required effect the needles must be inserted at the lowest part to which the dissection has been carried, that is to within a few millimetres of the ciliary margin. Further, it is essential to obtain the required effect at once by suitable adjustment of sutures, as, the operation being one which presupposes healing by first intention, any subsequent attempt at tightening will be futile. The sutures should necessarily be as fine as may be consistent

with strength; fine silk I found advocated by Hess, and have used, but prefer fishing gut for the sustaining sutures, and horsehair to bring together the edges of the wound. I think the sutures may advantageously be retained *in situ* for at least fourteen days, as they cause little inconvenience, and do not prevent the patient taking outdoor exercise.

The above constitutes the sum total of the modification I have found advisable in my experience of the operation, and with these limitations, I believe it to be thoroughly trustworthy and efficacious. The effects of the operation are, I think, in great measure permanent.

In the cases which are shown at this meeting, the elder, H. W—, now 18 years of age, was operated on three years ago, and he is now (with the exception of a slight loss of effect) as well as immediately after the operation (see Figs. 13 and 14). This was a case of unilateral ptosis (left).

In the case of the younger boy, J. P—, now 13 years old, the operation was performed consecutively on the right and left upper lids for double congenital ptosis. There was an interval between the two operations. One lid has obtained a somewhat less satisfactory curved furrow than the other, but the patient has quite got out of the craning attitude with which his efforts to keep his lids from covering his pupils was attended. Both of these patients have since obtained employment (from which they were debarred while the deformity existed) at Woolwich Arsenal and elsewhere.

The photographs exhibited (Figs. 13 and 14) indicate the elder patient before and after operation. As a point of interest as to closure of the lids subsequent to operations of this character, I have noted that patients can voluntarily close the lids in a perfectly normal manner; but in the case of one of my patients, at least, it was said that in natural sleep the eyelid which had been operated on was not perfectly closed.

Among the advantages that this operation possesses over others is that it leaves no deformity *per se*, as

Fig. 14.

Fig. 13.

Patient H. W—, before operation (Fig. 13), and three months after (Fig. 14).

the incision scar is soon concealed by growth of the hair of the eyebrow. Many of the older operations leave mementoes of their performance, even if they alleviate the original deformity. I owe my acquaintance with this operation to seeing it performed first in 1894, by the late Mr. Frank Hodges, of Leicester, whose untiring energy in acquiring new methods will be within the recollection of many members of this Society. I had the opportunity of performing the operation on two occasions in the following year. Having performed the operation altogether six times, I am inclined to think from the general measure of success attained, that it is one that should be more widely known and practised for what is an unsightly deformity. At the same time I would submit the modifications suggested to those surgeons who practise the operation.

(*May 2nd*, 1901.)

10. *Congenital fibro-vascular tumour of eyeball.*

By F. R. Cross.

(With Plate VI, figs. 3, 4, and 5.)

J. H—, a boy 10 weeks old, was brought to me on September 1st, 1892, with a large congenital fibro-vascular growth covering the outer half of the left eyeball. It occupied nearly half of the cornea (like a "dermoid"), spread outwards over the eye surface, and across the external canthus, which was involved in it, and terminated externally by a nodule of skin at the outer margin of the upper lid. The surface of the tumour was covered by the conjunctiva, thickened, vascular, and closely adherent to it; its deep attachment was in the episcleral tissue, and it passed widely upwards, downwards, and outwards, under the palpebral margin. The lids were quite free from the growth, excepting near the external canthus. The portion of the tumour exposed in the palpebral aperture

was full and very prominent, almost pendulous, and its pressure somewhat rotated the eyeball inwards. It was thought better not to operate at once.

The child was seen at long intervals. No change was observed in the relative appearances of the tumour, which was removed October 31st, 1899, when the patient was seven years old.

The corneal portion was first dissected up, and the free surface cauterised. Slitting the external canthus allowed of easier removal of the deeper portions of the tumour from the eyeball; it was nowhere encapsulated. Great care was taken not to injure the external rectus muscle, which was implicated. The conjunctiva, where free, was dissected back from the tumour, and afterwards used to cover the globe. Special pains were taken to avoid after-adhesion between the surface of the eyeball and the lids at the external canthus.

The patient is shown with coloured drawings of the condition before and after operation. The vertical diameter of the cornea of the affected eye is 1½ mm. wider than in the fellow eye.

Vision: R. $\frac{6}{6}$; L. $\frac{3}{60}$, J. 18. Field normal to nasal side, narrowed on temporal side in accordance with corneal scar.

(*Card specimen. June 13th*, 1901.)

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11. *Double coloboma of right lens.*

By A. Stanford Morton.

(With Plate VII, figs. 1 and 2.)

Jane T—, æt. 26 years, has noticed the right eye to be always defective. The vision is $\frac{1}{60}$ and J. 18 (the L. V. = J. 1 and $\frac{6}{6}$).

Externally the right eye looks the same as the left, except for a nearly central opacity in the lens, but on dilating the pupil the appearances indicated in the drawings become visible. Fig. 1 represents the full front view, and shows a coloboma of the lens down and out, with just a little shading of the lens above this at the margin of the pupil. On looking obliquely, however, as shown in fig. 2, a second much smaller coloboma becomes visible. There is no trace of suspensory ligament at either coloboma, nor is there any coloboma of iris or choroid. In both eyes there is a posterior staphyloma.

(*Card specimen. June 13th*, 1901.)

PLATE VII.

Illustrates Mr. A. Stanford Morton's case of Double Coloboma of Right Crystalline Lens (p. 179).

Fig. 1.

Fig. 2.

XII. COLOUR-BLINDNESS.

1. *A trichromic or three-unit case of colour-blindness.*

By F. W. Edridge-Green, M.D.

The patient, a man æt. 33 years, saw three colours in a bright solar spectrum—red, green, and violet. The orange and yellow he saw not as definite colours, but as mixtures of red and green. In the same way he regarded blue as a mixture of green and violet. No shortening of either end of the spectrum.

The following is a classification of the wools, silks, cards, and glasses belonging to my Classification Test. The name at the commencement was the epithet applied by him to each group:

Violet.—Violet, purple, blue-grey, blue, and blue-green (one card).

Blue.—Green and blue.

Green.—Green, blue, brown, and yellow.

Yellow.—Yellow, orange, greenish yellow, and yellow-green.

Pink.—Pink and purple.

Light red.—Orange, red, pink, and rose.

Red.—Red, rose, orange, and yellow-brown.

Red-brown.—Brown, grey, light green, and buff.

(Not named.)—Grey, brown, and black.

Tested with my Lantern Test.—Called yellow, "light red;" standard green, "blue;" purple, "violet;" and blue, "violet." (Rejected.) Named pure green, all neutrals, ground glass, ribbed glass, and all reds correctly.

Passed Holmgren's test without touching a confusion colour. He was examined and passed by Mr. Nettleship.

Read Stilling's coloured letters. Red and blue, 20 feet being normal at 33 feet 9 inches; green, 20 feet at 38 feet 2 inches; yellow, 20 feet, at 42 feet. He read all the letters on Stilling's pseudo-isochromatic plates.

The examination with the spectrum gave a key to his colour perception. He was never in doubt about red, green, and violet, and always named these colours correctly. Blue, yellow, and orange he said ought not to have names, and he was very uncertain about them. His manner when classifying these colours was characteristic of the indecision he felt regarding them. He would take a blue and put it with the violets, then move it away and put it with the greens. Finally, he put it in another class which contained as many greens as blues.

A man with this colour perception would be unsafe at sea, because he would be unable to distinguish the front light from the side lights. This is shown by the lantern test. A very interesting point in this case is that he had a much more acute sense for light of all colours than most normal-sighted persons. He was able to read Stilling's yellow letters on a black ground at more than twice the normal distance. It is evident, therefore, that quantitative tests, which assume that the colour-blind are short-sighted for colours, would fail to detect a case of this kind.

(*Card specimen. October 13th*, 1900.)

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2. *The evolution of the colour-sense.*

By F. W. Edridge-Green, M.D.

In two papers, written in 1871 and 1872, Lazare Geiger came to the conclusion from an examination of the Bible, the Védas, the Zend-Avesta, and the Homeric poems, that

the people of those times were not able to distinguish all the colours which are visible to us. Hugo Magnus, Professor of ophthalmology at Breslau, in two very able papers, brought forward a great number of facts to prove that the sense of light was developed first, and then the sense of colour. He propounded a theory that the sense of colours was developed in the order of the refrangibility of the latter—that is to say, that red was seen first, then orange, then yellow, then green, then blue, and lastly, violet.

In an article in the *Nineteenth Century* for 1877, Mr. Gladstone adopted Magnus's theory, but inclined to the opinion that Homer's colour vision was even more restricted than Magnus thought. He thought that red was the only colour definitely seen by Homer. On reading Mr. Gladstone's article it will be seen that many points of difficulty in the acceptance of the theory occurred to him. The opponents of Magnus's theory at once pointed out a weak point. This was that it did not give any sufficient reason for red being seen before the other colours, as light of all degrees of refrangibility had always been acting upon the eye. It was also definitely proved that violet was seen by the ancients, as a visit to the British Museum will at once show. The colour which is seen corresponds as nearly as possible to spectral violet. It occurred to me that perhaps the facts of colour-blindness might throw some light on the question. I find that not only does all the evidence support the opinion that the sense of light was developed first, but that in the development of the colour-sense all the stages of psychophysical colour-blindness were passed through, until the present stage of colour-vision was reached.

On examining the facts which can be obtained as to the colour-sense of the ancients, I was at once struck by the extraordinary resemblance to cases of colour-blindness, and the older descriptions correspond to the most marked cases. On examining museums we find certain colours used, and in literature certain colour-names employed, and

great confusion between many colours. But the mistakes made are always those of the colour-blind, and when we have estimated to which class of the colour-blind a particular author belongs, we find that we have a perfectly intelligible explanation of the colour-names of that author.

I found that an examination with the spectrum gives a key to the colour-perception of a colour-blind person, and in the same way I found the description of the rainbow by different authors gives a key to the colour-perceptions of those authors. We can understand a normal-sighted person saying that in the rainbow there were innumerable colours, but we cannot comprehend how he can describe it as monochromatic, or as consisting of only two or three colours. Many ancient authors describe the rainbow as tricoloured in exactly the same way as the trichromic do at the present time. Aristotle describes the rainbow very carefully; he says that ordinarily it consists of three colours—red, green, and violet, but when exceptionally brilliant, an indication of a fourth colour, yellow, can be seen between the red and the green. The tetrachromic describe it in the same way.

Eleven years ago I read a paper before this Society on a "New Theory of Colour Perception," and I find that this theory is in perfect accordance with, and gives an easy explanation of, the evolution of the colour-sense.

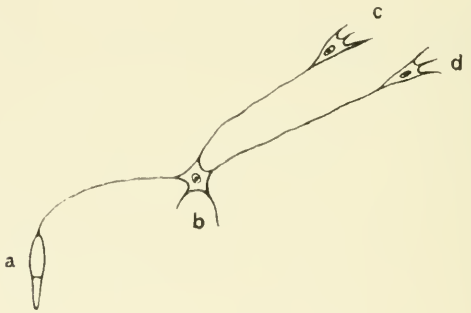
I will give a short outline of this theory. In addition to being supported by anatomical facts, it explains all the extremely complicated phenomena of sight, including those facts which are confessedly incompatible with any other theory. I have assumed that the cones of the retina are insensitive to light, just as, in fact, the optic nerve-fibres themselves must be. I regard the cones simply as the terminations of the optic nerve-fibres in the retina. This hypothesis at once disposes of the main objection which made Kühne say that the visual purple could not be essential to vision, and could not be the visual substance. According to this hypothesis, light falling upon the retina liberates the visual purple (rose) from the rods, just as heat melts an ointment,

and a photograph is formed, the cones conveying the impression of the photograph to the brain. It will be seen, therefore, that the cones are only sensitive to chemical changes in the visual purple, and not to light itself in the sense of conveying any light sensation to the brain. They are sensitive in one way, namely, that when light falls upon them it causes the inner limbs to contract, but this change has been noted when light falls on the other eye, or on the skin. This may be one of the ways by which the visual purple is drawn out, and support is lent to this view by the fact that my experiments on the disappearance of lights can be best made with only one eye, and when no light is allowed to fall on any part of the body with the exception of that which falls on the fovea centralis. In darkness, therefore, the visual purple is in the rods, and the yellow spot of the retina corresponds to the sensitive plate of a photographic apparatus, which is prepared by light immediately it is required for use. On waking from sleep in the middle of the night the field of vision often appears absolutely dark for some seconds, although there may be sufficient light by which to recognise objects. I have noted that just preceding vision a number of rose-coloured circles appear in the centre of the field, and round this centre a great number of rose-coloured and green circles which interlaced, and appeared to be rapidly moving, the appearance being very similar to the subjective sensation caused by intermittent light. I have made numerous experiments, which show that light may fall on the fovea centralis without producing any sensation.* It is easy to suppose that light acting on the visual purple effects chemical changes in it, that these chemical changes excite the ends of the cones, and a sensation of light is conveyed to the brain. Fig. 15 shows the connection of a cone of the retina, *a*, with a ganglion cell, *b*, of the centre for visual memory; *c* is a cell of the colour-perceiving centre, *d* a cell of the form-perceiving centre.

* *Ophthal. Review*, London, October, 1900.

It is not at all necessary that we should assume that there are any special substances in the retina, such as the hypothetical substances of the retinal theories of colour-vision. It is easy to suppose that light in decomposing the visual purple gives rise to nerve impulses which differ in character ; for instance, wave length, just as the physical light waves differ themselves in wave length. A varying degree of acidity would be almost sufficient for our purpose. Therefore, in the impulse itself we have the physiological basis of light, and in the quality of the

Fig. 15.



impulse the physiological basis of colour. It will be seen from this basis that there is no reason why we should not see an innumerable series of colours. But this is not the case either with the colour or any other perceptive faculty. A very simple experiment will suffice to demonstrate this point. All that is needed are two lights, a screen, and an opaque object. Let a equal the light thrown on the screen by the light A, and b that thrown by the light B. The portions of the screen free from shadows receive $a + b$, whilst the shadows receive light only from one source. It is, however, easy to adjust the lights so that one shadow is quite invisible. The same applies to colour.

It will be seen that the cerebral cell conveys to the mind information concerning the physical stimuli within the power of perceiving differences possessed by that cell.

It is obvious that the psycho-physical theory could be applied in another way, that is, we could assume that the nerve impulses caused by a certain colour differed from other nerve impulses in a different manner from the range of the spectrum; for instance, that yellow and blue differed more than any other two colours. But the facts show that the simplest form of the psycho-physical theory is true – that is, that the nerve impulses differ in accordance with the physical light series.

The physical series of colour.—It is evident that before we can have a psycho-physical series (that is a physical series as it appears to the mind), we must have a physical series; and this is admirably represented in the case of colour by the solar spectrum. This is the most perfect example of a physical series which can be obtained. By dispersion the sun's light is spread out in the form of a series, the wave lengths of the units gradually diminishing from the red to the violet. The presence of gaps in the solar spectrum in the shape of Fräunhofer's lines shows that the spectrum is pure, and that the rays of light do not overlap.

The points of greatest difference may be obtained by measurement of the wave lengths. It is evident that the two points of greatest difference do not come within the visual range at all, because the waves in the infra-red present a greater physical contrast to those in the ultra-violet than any two rays in the visible spectrum. If we take the portion of the physical series represented by the spectrum, the two points of greatest physical difference are the first visible ray of red, and the last visible ray of violet. The third point of greatest physical difference will be the centre of the spectrum. The next two points will be midway between the third point of difference and the ends of the spectrum. These and subsequent points can be found with the aid of mathematics.

The psycho-physical series of colour.—When a physical series has been obtained, the mental impression of this series constitutes a psycho-physical series. The appearance

of the spectrum to any person constitutes the psycho-physical colour series for that person. The question then is, what is the appearance of the spectrum to the majority of persons? Most persons say that they can see six definite colours in the spectrum—red, orange, yellow, green, blue, and violet, and that one colour appears to shade off into those adjacent to it. We can examine the spectrum in another way—namely, by observing only a small portion at a time, either using shutters to a spectroscope, or letting the spectrum pass through a slit, which allows only a small portion to pass. When the spectrum is viewed in this way, it appears to be made up of a series of monochromatic bands. The size of these monochromatic bands differs with different persons; that is, a band which is monochromatic to one person is not necessarily monochromatic to another. These bands are absolutely monochromatic; *i. e.* if a portion of green were taken, the observer could not say which was the yellow and which was the blue side of the portion of light shown. But we know that the portion of light, though apparently monochromatic, contains rays of light which differ very considerably in wave-length; therefore we have a number of physical units which cannot be distinguished from each other. These units are seen under the most favourable circumstances for the detection of any difference, the adjacent colours being excluded, and yet they appear alike. The first obvious inference to be drawn from this is, that the rays of light occupying a monochromatic band are identical for the observer as far as perception is concerned. In the psycho-physical colour series, therefore, the absolute psycho-physical colour units are portions of the spectrum which appear monochromatic when the remainder of the spectrum is shut off. For example, take the several varieties of green which we can distinguish. As the band of colour appears monochromatic, we could substitute an equal number of any one of the rays of light entering into its formation without altering its appearance as far as colour is concerned. From this it is evident that nearly all the

experiments which have been made by physicists with regard to colour will have to be looked at from a psycho-physical standpoint, as this is a source of error which appears to have been very generally overlooked. As we can only tell an absolute psycho-physical colour unit from the adjacent units by carefully comparing them, it is evident that the difference between them is not sufficient for practical purposes, so we come to the approximate psycho-physical colour units—*i. e.* colours which can be easily distinguished from each otherwithout comparison.

These approximate psycho-physical units for a normal-sighted person are six in number—red, orange, yellow, green, blue, and violet.

Fig. 16 represents a spectrum as seen by a normal-sighted person. A and B represents the centre points of approximate psycho-physical units—green and blue; that is,

FIG. 16.

colours corresponding to these portions of the spectrum would be easily distinguished without comparison. The rays included between A and C form an absolute psycho-physical unit; that is, the portion included between A and C appears monochromatic. If shutters were used to the eye-piece of the spectroscope, the observer would not be able to say which was the yellow and which was the blue side of the portion of light shown. If, however, he were shown the coloured band from A to D, he would be able to distinguish between the colour at A and that at D. But if he were shown a colour corresponding to D, and were asked to which portion of the spectrum it corresponded without being allowed to compare it with other colours, he would find great difficulty in indicating the

right position. There are, therefore, six definite points of difference in the spectrum to a normal-sighted observer, corresponding to the centre of each of the colours—red, orange, yellow, green, blue, and violet. As each of the colours at these points must, by the presence of the adjacent points blend with each other, we have the approximate psycho-physical units formed. It will be seen that if the size of the absolute psycho-physical units, as A C, be increased, the size of the approximate units will also be increased. As the spectrum remains the same length, the increase in size of the approximate units results in their re-arrangement and diminution in number, causing colour-blindness: first, five instead of six distinct points are seen in the spectrum, in the next degree four, and so on, until total colour-blindness is reached.

I have classified the colour-blind in accordance with the number of colours which they see in the spectrum. If the normal sighted be designated hexachromic, those who see five colours may be called pentachromic, those who see four tetrachromic, those who see three trichromic, those who see two dichromic, and the totally colour-blind monochromic.* An examination with the spectrum gives a key to the colour-perception of any person. It will be noticed that the colours appear in a definite order, and exactly as we should theoretically expect, at the points of greatest difference. It is obvious that the three points of greatest difference are the centre and ends of the spectrum. Where will the fourth point of difference be situated? If the units of the physical series differed from each other in a proportional manner, the fourth and fifth points of difference would appear at the same time and be situated at points midway between the centre and ends of the series. The units of light do not, however, differ from each other in a proportional manner, therefore the fourth point of difference will appear before the fifth. The waves of light at the red end of the spectrum are larger than those at the

* *Colour-blindness and Colour Perception*, International Scien. Series.

violet end, therefore the fourth point of difference will appear at a point midway between the red and the centre of the green, namely, the yellow. An example with smaller numbers will show why the fourth point of difference should appear on the red side of the green. Let us suppose that we have a series of vibrating members, the vibrations being from 5 per second for the lowest member to 105 per second for the highest member. There will be much more difference between the first and second members of the series than between the last member and the one just before it. In the first case there will be a difference of $\frac{1}{5}$, in the second case $\frac{1}{104}$, a very much smaller fraction. The fifth point of difference will appear midway between the violet and the centre of the green. The sixth point of difference will appear on the red side of the fourth point. There will then be two points of difference between the red and the centre of the green, namely, orange and yellow. It is evident that when orange is seen, the fourth point of difference, yellow, will appear to have moved more towards the green, the fourth point of difference now being a combination of the two, namely orange-yellow. This can be seen to take place if a spectroscope be arranged so that no orange can be seen. A spectrum of this kind can be obtained with a fine slit; on slightly widening the slit, so that more light is admitted, the orange will be seen, and the yellow will appear to change its position and move towards the green. The seventh point of difference will appear between the green and violet; that is to say, there will be two points of difference or colours seen between the green and the violet instead of one.

It is curious to note that those possessing a lower degree of colour perception often invent colour terms which are not intelligible to the normal sighted. Thus, those possessing trichromic vision often talk about red-green when they mean yellow, and violet-green when they mean blue, and declare that these terms are more correct definitions of the colours. When there is a shortened spectrum, as we should theoretically expect, all the centres of the colours are moved

towards the unshortened side. This in the dichromic has led to the division into red and green blind. It is obvious that when the red end of the spectrum is shortened the centre of the green will no longer be the centre of the spectrum, and the neutral point will be proportionately nearer the violet end. Those who see seven colours in the spectrum have an exceptionally good colour-perception. They can match colours with greater ease, and have a better memory for colours than the normal-sighted. They can also recognise differences of colour which are not perceptible to the normal sighted. In testing for those employments on sea and land which require a good colour perception, I class those who are pentachromic and tetrachromic with the normal-sighted. I may mention that the ordinary tests which are employed by railway companies and the mercantile marine allow those who are trichromic to pass as well, but this is not right, as those who are trichromic are always in difficulty over yellow, although they distinguish red from green easily enough.

Cases of colour-blindness may be divided into two classes which are quite separate and distinct from each other, although both may be present in the same person. In the first class there is light as well as colour loss. In the second class the perception of light is the same as the normal-sighted, but there is a defect in the perception of colour. In the first class certain rays are either not perceived at all or perceived very imperfectly. Both these classes are represented by analogous condition in the perception of sounds. The first class of the colour-blind are represented by those who are unable to hear very high or very low notes. The second class are represented by those who possess what is commonly called a defective musical ear. It is with the second class that we are specially concerned in this paper. Colour-blind individuals belonging to this class can be arranged in a series. At one end of this series are the normal-sighted, and at the other the totally colour-blind. Let us see whether the degree of colour-blindness which just precedes the totally

colour-blind will give us an idea of the perception of colour when the colour sense was first developed. I had an exceptional opportunity of studying a case of this kind, as the patient was colour-blind with one eye. It is an interesting fact that the colour-blind eye was much the better of the two, as he could see fine lines in the spectrum with this eye which were not visible with the other. On examining him with the spectroscope, I found that he saw the two ends of the spectrum tinged with colour, and the remainder grey. Neither the red nor the violet appeared of the nature of a primary colour, but gave the impression that they were largely diluted with grey. The red commenced at the B line of Fraünhofer. He could see A as a blacker line on a grey ground. The red practically ended at C, and the band of colour was uniform in intensity, and did not vary like this portion of the spectrum did to the left eye.

It will be seen that the colour-perception of his right eye was limited to two colours, namely, those which present the greatest physical contrast to each other, red being produced by the largest waves, and violet by the smallest. This is exactly what we should expect, it being most probable that those physical stimuli which were most dissimilar would first be perceived as different. The examination with the spectroscope gave a key to his colour-perception. Tested with a lantern at a distance of fifteen feet, he recognised the red glass at once. Tested with pure green, blue-green, blue, yellow, and various depths of neutral glass, he declared uniformly that they had no colour, but remarked that I had not shown him a violet glass.

Let us now compare this case with the facts concerning the evolution of the colour-sense which can be obtained from the examination of museums or literature. We find that the two oldest European colour terms are those which are represented by the red and violet of the spectrum. We find that nearly all the European colour terms for red have had a similar root, and that all the European colour

terms for violet have had a similar root. Homer's colour vision was dichromic in the sense of the case that I have just described. He saw red and violet, and correctly used the epithets corresponding to these colours.

The following are the derivations of the ordinary colour names :

Red.—Old English : red, reed, read. Anglo-Saxon : reád, reód ; Old Saxon : rōd. Old Friesic : rād. Dutch : rood. German : roth, rōt. Old High German : rot. Swedish and Danish : röd. Icelandic : rawdr, rjodr. Gothic : ráuds. Welsh : rhudd. Armorican : rnz. Irish and Gaelic : ruadh. Latin : ruber, rufus. Greek : ἐρυθρός. Sanskrit : rudhira, rohita.

Orange.—There is no old colour word corresponding to orange.

Yellow.—Old English : yelow, yelwe. Anglo-Saxon : geolu. Dutch : geel. Old Saxon and Old High German : gelo. German : gelb. Icelandic : gulr. Swedish : gul. Danish : guul. Fisk gives the Indo-Germanic root of the above as zhelo, to be green or yellow. It is impossible to find a word in the ancient languages to correspond to yellow. The best are :—Latin : helvus, light bay. Greek : χλόη, young verdure ; χλωρός, greenish yellow. Sanskrit : hari, tawny. Ξανθός has also been said to represent yellow, but has been used for all the colours from scarlet to blue-green.

Green.—Old English : grene. Anglo-Saxon : grēne. Dutch : groen. Old Saxon : grōni. Old High German : groni. German : grün. Danish and Swedish : grön. Icelandic : graenn. All these are from the root of green. French : vert. Latin : viridis. The old Greek words for green cannot be distinguished from those for yellow and blue.

Blue.—Old English : bla, blo, blew, blue, livid, black, from Icelandic : blār, livid. Danish : blaa, blue. Swedish : blå. Dutch : blauw. Old High German : blāo. German : blau. French : bleu. There is no ancient word corresponding to blue, and even in recent times the same word

has been used for both blue and green; for instance, the Welsh word glas was used for blue, green, and grey.

Violet.—French: violette. Old French: viole. German: violet. Latin: viola. Greek: ἴον. Here we have, like red, a colour epithet the derivation of which can be traced back to the time of Homer. The violet is one of the very few flowers mentioned by Homer, and he used the name of the flower as a colour epithet, and applied it correctly, and to the same class of objects as πορφύρεος.

Purple.—Old English: purpre, pourpre. Old French: purpre, porpre, pourpre. French: pourpre. German: purpur. Latin: purpura. Greek: πορφύρεος. Again we have a colour epithet of very ancient origin. There has been a good deal of dispute as to the meaning of πορφύρεος, but if we assume that it represents spectral violet, we shall have an intelligible explanation of its use in olden times. Even now the word purple is applied by many authors to describe the violet of the spectrum. In olden times Xenophanes used πορφύρεον to describe the third and cold colour of the rainbow.

The chief argument used by the opponents of the historical evolution of the colour-sense is that certain colours were used by the ancients, and that, therefore, this proves that they could not possibly have had a defective colour-perception; but because they used these colours it does not follow that they actually saw them as we see them. As a matter of fact, it is curious to note how very rarely colours other than the fundamental colours that they saw were used. For instance, on examining the earliest known paintings (after the monochromatic stage) and coloured objects, we observe a constant repetition of red and violet, and many mistakes are made with the other colours, which are rarely used. It will be noticed in the British Museum that in one mural painting several horses are represented, some are white, some are black, some are red, and some are green. In all probability the artist meant to represent a brown horse, and made one of the commonest mistakes of the colour-blind. I also find that the faces of

many of the people are painted green, and a confusion between blue and green in later paintings is very common.

I have a series of paintings by colour-blind persons, and the mistakes made are similar to those which I find in museums in the work of the ancients. The blunders of those who are most colour blind are to be found in the oldest paintings.

It will be noticed that not only is the theory in accordance with facts, but the difficulty which prevented the acceptance of the older theory is overcome. If it be assumed that the sense of light was developed first, and that a certain portion of the visual centre was set apart for the perception of colour, when it was very small and ill-developed it would only be able to perceive differences of considerable magnitude; but as more cells were added to the centre greater and greater power of perceiving differences arose. Each nerve-cell might at first have been connected with many optic nerve-fibres, and the number gradually diminished as evolution proceeded. The spectrum forms a series, the ends of which present the greatest physical contrast to each other, the red waves being the largest, and the violet the smallest; therefore, assuming that the nerve impulses differ in a similar manner, red and violet were the first two colours seen. The spectrum was therefore first seen as grey, then with a tinge of red at one end, and violet at the other. As the colour sense developed it was not necessary that the rays should be so far apart before a difference was seen, and so the grey band gradually diminished until it entirely disappeared, and the two colours met in the centre of the spectrum. A third colour, green, then appeared at the central point; then yellow appeared, making four colours; then blue, making five; and finally, orange, making the normal six. In some persons evolution has proceeded further, and seven colours are seen, there being two colours between the green and the violet instead of one. Those who see seven colours in the spectrum are very few

in number, and possess an unusually good colour perception. They have a better memory for colours, and recognise differences in colour which are not evident to other people. It is curious to note that we do not possess a definite colour name for the seventh colour. Newton called it indigo, but the ordinary indigo is a greenish blue. The powdered indigo in a raw state, however, is a violet blue, which correctly describes the colour. It is not necessary to consider the further evolution of the colour sense, as I have not met with a person who could see more than seven colours in the spectrum; but we know how evolution will proceed, the extra colour appearing first on the red side of the green, then on the violet side.

It will therefore be seen that colour-blindness is only an example of an earlier state in the development of the colour-perceiving centre.

One of the most disputed points is the number of colours that can be seen in the spectrum. One writer declares that four is the number of colours, and accuses Newton of a lurking disposition to mysticism because he says there are seven; another says five is the correct number; another six; and another seven, declaring that Newton was right. Others, again, claim that the number of colours is innumerable. It is very easy to limit the number of colours, and as these colours must blend with other colours, we have many hues included under one colour. But it is ridiculous to assert that yellow-green is a definite colour; its component parts of green and yellow are plainly evident, which is not the case with the primary colours; for instance, yellow has no intrinsic relation to either orange or green. It will be noticed that the colours have a value which corresponds to the order of evolution; thus orange is the one which is the least readily recognised as a definite colour, then blue, and so on backwards to red and violet.

Unfortunately the subject of colour-blindness has been studied almost entirely from a theoretical standpoint, and so a great many of the facts have been overlooked. The following will give the normal-sighted the best idea of the

vision of the colour-blind. He knows that he can see six definite colours. Let him for the five-unit imagine that five of his adjacent colours are spread over the spectrum, for the four-unit four, for the three-unit three, for the two-unit two, and for the one-unit one. Looking at the subject in this way, he will at once comprehend how it is that the colour-blind are able to recognise colours, and correctly to name them. As an example of this method of looking at the subject, let us represent the two-unit by the two adjacent units, blue and green. It is obvious that a normal-sighted person could distinguish different colours, even if only two were visible to him. Then colours might be named in the following way. A dark but pure green would represent red; a very bright green, orange; the brightest possible green, yellow; a bright but not quite so pure green, yellow-green; a duller and not so pure green, pure green; a mixture of the two colours, a blue-green; a blue with a trace of green, blue; the purest possible blue, violet. This is how a certain two-unit learned to distinguish between colours, and his friends had great difficulty in making him name them wrongly.

It is probable that although we have gained in colour-perception we have lost in acuteness of sight. It is well known that savages have a far more acute sight than is normal in civilised communities. I showed before this Society a colour-blind person who was able to read test types of all colours at a distance far beyond the normal. The sense of colour and the perception of light and shade are quite distinct. In the same way, acuteness of hearing and the power of saying whether two notes are of the same pitch, are not related. It is easy to suppose that primitive man saw all objects of a uniform hue, just as they appear in a photograph; but that he had a very acute perception for differences of luminosity. In course of time a new faculty of the mind, a colour-perceiving centre, became developed. This colour-perceiving centre in its undeveloped state was first able only to appreciate those differences which were caused by the waves of light which are physically most

different. Evolution then proceeded on the lines that I have already indicated. (*March 14th*, 1901.)

The PRESIDENT said a few years ago he formed one of a deputation to the President of the Board of Trade, whose object was to direct attention to the imperfection of the tests used for railway servants. The matter was presented as simply as possible, but even that seemed much too complicated for the Board of Trade. If they had used a diagrammatic representation, such as Dr. Edridge-Green had shown, he feared it would have seemed more complex. He felt sure that those who failed to pass the tests mentioned by Dr. Edridge-Green should not be employed as public servants.

Mr. HERBERT PARSONS reminded Dr. Edridge-Green of a paper by Professor Haycroft, read before the Royal Society several years ago, in which that gentleman propounded the same theory, white light being the primitive sensation. It was discussed by Lord Rayleigh from the physical standpoint and by Professor Foster from the physiological.

Dr. EDRIDGE-GREEN, in reply, said he had read the paper referred to, but it came twenty years after any of those he had mentioned, and only supported the primary hypothesis that the sense of light was developed first. With regard to the President's remarks, he (Dr. Edridge-Green) thought the Board of Trade had been unjustly criticised. When he first read his paper the President of the Board of Trade asked him to advise the Board on the question. He took a great deal of trouble over it, and brought out his book in the International Scientific Series. The results of his examinations showed that Holmgren's test was inefficient. The Royal Society, however, appointed a Committee which upheld Holmgren's test. The Board of Trade, however, knew from the facts he had brought forward that the Holmgren test was unsatisfactory, a point generally recognised by specialists at the present time, even by those who had previously supported it most strongly.

XIII. HISTOLOGY.

Sections showing opaque nerve-fibres in the retina.

By Percy Flemming.

The section shows that the nerve-fibres lose their myeline sheaths abruptly at the posterior end of the lamina cribrosa, and regain them at irregular levels at the anterior end. The fibres show many varicosities—some globular, others spindle-shaped.

The fundus had been examined two days before death, and the existence of a white patch, having the usual characters of opaque nerve-fibres, had been noted.

(*Card specimen. March 14th*, 1901.)

XIV. PATHOLOGICAL ANATOMY.

1. *Microscopical specimen of an osteosarcoma of the orbit.*

By John Griffith.

The tumour was removed from the left orbit of a labourer, æt. 29 years, on December 2nd, 1899. He first came under my care at the Royal Westminster Ophthalmic Hospital in June, 1899, with a history that he had had pain at the back of the eye for three weeks. There was very little noticeable then:—slight proptosis, limited movement upwards with diplopia, and pain on looking to the right or left. The left optic disc was congested. R. V. = $\frac{6}{6}$. L. V. = $\frac{6}{18}$ + 2·5 D. sph. = $\frac{6}{12}$. A small nodular mass was felt on roof of orbit towards the nasal side. No history of injury, of syphilis, or of tubercle.

He was placed on ascending doses of iodide of potassium, but as the symptoms did not improve and the growth increased in size, an exploration of the orbit was made by a curvilinear incision through the brow on July 19th. Nothing could be discovered beyond some soft cancellous bone on roof of orbit near its margin. This was regarded as carious bone, the result of periostitis, and was gouged away. There was a temporary improvement in his neuralgic pains, and the eye seemed to be less prominent, and to move better. In October the pain returned, the eye became more prominent, and there was numbness with anæsthesia along the course of the supra-orbital nerve. Bromide and iodide of potassium were prescribed without much benefit. He begged me to take the eye out. After a consultation with my colleague, Mr. Work Dodd, I

decided to exenterate the orbit. This was carried out on December 2nd. The whole roof of the orbit, extending to the optic foramen, was involved in a soft, cancellous, bony growth, which crumbled away under pressure of the finger. The eyeball and the orbital contents were removed, and the tumour gouged away without penetrating the cranial cavity. The orbital cavity granulated up, and he was discharged on December 23rd. From that date I lost sight of him, and learn that he died on April 2nd, this year, when I was absent from town.

Dr. England, who attended him in his last illness, wrote that the tumour returned in his orbit, involved the nose, from which escaped a bloody discharge, and he became very emaciated and delirious. He died suddenly. Dr. England ventured the opinion that the cause of death was asphyxia, due to the invasion of the pharynx by the growth. No necropsy was made.

The microscope reveals a tumour consisting of cancellous bone, the meshwork of which is occupied by myxomatous tissue, *i. e.*, a tissue composed of branching, stellate cells with deeply-staining nuclei. There are no definite cells of a sarcomatous nature in the sections examined, but the malignancy of the growth points in that direction.

(*November 8th*, 1900.)

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2. *Hypertrophy and ossification of tarsus.*

By Major H. Herbert, I.M.S., Bombay.

I wish to report this case, because in the limited literature at my command there is no mention of anything similar.

The patient, a male Hindu, æt. 33 years, came on account of a persistent conjunctivitis of the right eye, with mucopurulent discharge. The upper lid could not be everted.

The tarsus, although not greatly thickened, was hard and rigid, moulded to the curve of the eyeball, and extended upwards and backwards into the orbit, especially about its outer end. Here its depth, measured from the lid margin, was about three quarters of an inch; the thickened upper margin could just be felt by the finger pressed back into the orbit when the patient looked down. But the enlargement rapidly diminished inwards, and the inner fourth or less of the tarsus appeared normal. Looking up at the conjunctival surface of the lid, pulled away from the eyeball, a small opening was seen about the middle of the tarsus, exposing a piece of dead bone, which was extracted. The presence of this bare bone doubtless explained the conjunctivitis. An enlargement upwards of the tarsus of the left upper lid, quite similar in shape and extent, was found; but it appeared thinner and was pliable, bending sufficiently to allow of ordinary eversion of the lid. Both palpebral apertures were narrowed, and in such a way that the widest part was about the junction of inner and middle thirds. There was a firm, painless, pre-auricular, subcutaneous nodule on the right side, probably glandular; although, when cut down upon, nothing was found superficial to the parotid. The right tarsus was exposed through a transverse skin incision, and found to be largely bony, not so much toward the lid margin as in the upward extension. A fair amount of the ossified tarsus was removed, with some sacrifice of conjunctiva, but no attempt was made to reach the upper margin. Sections of the decalcified tissue show bone formed and forming in membrane, in parts dense, in parts fairly open, cancellous. A small piece of the upper part of the left tarsus was excised, and found to consist of fibrous tissue only. The conjunctiva of this side was almost normal. There was an old corneal opacity of the right eye.

(*March 14th*, 1901.)

3. *An encapsulated sarcoma of the right orbit.*

By Simeon Snell.

W. H—, æt. 36 years, a miner, was admitted into the Sheffield Royal Infirmary, under my care, on December 7th, 1900. He sought advice as to a swelling in the right lower eyelid. This was first noticed by him as a small lump five or six months before. It appears to have grown more rapidly during the last few weeks. At no time has it occasioned him pain. On examination a prominence is noticed in the centre of the lower eyelid. On palpation this is discovered to be due to a very freely moveable tumour, which is tense, and fairly smooth on the surface. Pressure on it makes it recede along the floor of the orbit, and on inserting a finger on the inner side of the lower eyelid it can be readily pressed forward. It was then noticed to be fully the size of a pigeon's egg. There is no pain or tenderness, nor is there redness of the surface of the eyelid, nor are there any enlarged glands. The eyeball is a little pushed up, and lies on a plane superior, and apparently posterior, to that of the left globe. Vision R. $= \frac{6}{4}$, L. $= \frac{6}{3}$.

On December 10th, 1900, ether was administered, and the tumour removed. An incision one and a half inches long was made in the fold on the outer surface of the lower eyelid, and above the edge of the orbit. A little dissection revealed the tumour, which was found to shell out easily. It had no pedicle, and there were only loose connective-tissue attachments. It was about the size of a pigeon's egg, was constricted across the middle, which gave it somewhat the shape of a kidney, and was of firm consistence. I am indebted to Mr. Treacher Collins for kindly examining the tumour. I append his report.

(*Card specimen. May 2nd*, 1901.)

Report of pathological examination of the tumour removed from the right orbit of Mr. Snell's case—W. H—.

The growth, which has been preserved in formol, is encapsuled, and somewhat the shape of a kidney. It measures 3 cm. in its longest diameter, and 2 cm. transversely. It is slightly concave on one surface, and convex on the other. One border is regularly convex, and the other dimpled like the hiatus of a kidney. There are three furrows on the convex surface, one rather deep one. The capsule of the growth shows no signs anywhere of having been cut into at the operation.

On section, the growth is found to be of very firm consistency, and to have the appearance of an unripe pear. Microscopical examination shows it to be a large spindle-celled sarcoma. Groups of the spindle-cells are in places cut transversely, and appear like collections of round-cells.

January 15th, 1901. E. Treacher Collins.

4. *Case of carcinoma of the corneo-scleral margin.*

By Leslie Buchanan, M.D.

J. S—, æt. 60 years, a much broken-down looking man, came to the Glasgow Eye Infirmary on the 10th May, complaining of pain in the left eye, extending into the left side of the head, and of a growth beside the cornea at the outer side.

The history of the condition was that eight weeks before he first experienced discomfort in this eye. Gradually the discomfort developed into pain, which was not severe, and was only occasional. A fortnight later he first noticed the

presence of a small pink spot on the white of the eye. Since then the pain became more severe in character and more constant in occurrence.

Present condition.—There is a growth under the conjunctiva at the outer side of the cornea, slightly below the horizontal meridian. The mass is oval in shape, and flattened on its surface, whilst it is slightly lobulated. The colour is deep pink, and the consistence is moderately soft; the tumour measures 6 mm. in its greatest length, which is situated radially, by 3 to 4 mm. in breadth, by 2 to 3 mm. in thickness. The growth is apparently fixed at the corneo-scleral margin, but moves over the sclerotic at its outer extremity. It only overlaps the cornea by about 1 mm. Several distended vessels course under the conjunctiva to the growth from above and below. There is no black pigment in the tumour, only a very small spot being seen just at the corneal margin at the lower part of the mass.

The tumour was removed freely, both sclerotic and cornea being incised in doing so, and the wound scraped. The wound healed well, and recovery was rapid, the pain entirely disappearing.

Histological examination showed that the growth was composed of epithelial tissue in somewhat gland-like tightly compressed masses, and that it was covered by conjunctival epithelium, which, although considerably thinned, was intact. The growth was not freely vascular, and there was little connective tissue between the lobules of which it was composed.

Remarks.—The tumour was considered to be carcinoma from the beginning, as it occurred in an old man, was not pigmented, was soft in consistence, and pink in colour. The freedom of the conjunctiva seemed to indicate that the growth was not epithelioma of the squamous type. The possibility of the growth being a gumma was rejected, as the mass was moveable, and was not accompanied by inflammatory signs in the conjunctiva. For similar reasons the growth was not at all like tubercle. The rapidity of

the growth of the tumour seemed to indicate that the mass was malignant in nature.

The eye was not removed, because the growth was still moveable over most of its extent, and there seemed to be a possibility of getting below it by excising the outer layers of the cornea and sclerotic below the mass.

(*Card specimen. June 13th*, 1901.)

5. *Note on a streptothrix mass (actinomyces?) removed from the right inferior canaliculus.*

By Geo. Mackay, M.D.

From the right inferior canaliculus of an elderly lady I recently removed, after partial slitting, some dull, grey-green masses of a soft concretion which had greatly distended the lumen without apparently invading the surrounding tissue. She stated that a lump had been forming for several months, but had only recently begun to inflame and discharge a little pus through the punctum. This slight reaction soon subsided after evacuation of the contents, and I should have regarded the case as an ordinary leptothrix concretion had not my friend, Dr. D. Noël Paton, the Superintendent of the Royal College of Physicians' Laboratory, suggested that the microscopic appearance of a fragment of the fungus, which I sent him, closely resembled actinomyces.

Dr. Welsh, Pathologist to the Edinburgh Royal Infirmary, has kindly made a film preparation by Gram's method, and a section stained by the same method, which show well the features of the growth under high magnification.

I submit also two photomicrographs, the one showing the branching filaments of the mycelium, the other the rosettes or arcs of club-shaped bodies, resembling colonies

of a ray-fungus, with the mycelium extending in a felt-work all round.

Mr. Foulerton, of the Middlesex Hospital, who, with Dr. Price Jones, has recently added to our knowledge of these fungi, has kindly favoured me with his opinion, that the fungus "is a typical streptothrix, which is just beginning to shew the process of chain sporulation."

The *branching* of the filaments, the retention of Gram's stain, and the formation of bulbous swellings at the ends of some of the filaments, differentiate the growth from leptothrix, and justify its inclusion among the streptothriceæ, and possibly the actinomyces. Unfortunately, no cultures were made, and it is therefore very difficult to identify the species; but I should value the opinion of any member of the Society interested in these lowly organisms.

(*Card specimen. June 13th*, 1901.)

The PRESIDENT said most of the members must have removed similar masses, and his experience was that there was one most important diagnostic sign, namely, a little bead of pus at the entrance of the swollen canaliculus.

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6. *Cyclitis: a study of inflammatory exudations into the vitreous body in cases of cyclitis.*

By LESLIE BUCHANAN, M.D.

(With Plates VIII to XVI.)

CYCLITIS has been chosen as the subject of critical investigation, as it appeared to be of importance both from a clinical and a pathological point of view, and as it seemed probable that a more exact knowledge of the nature of the process would greatly assist in the framing of a prognosis.

In order that even fairly trustworthy results might be obtained, a large number of cases have been investigated, and an average of these cases has been drawn. By this

means it is hoped that truth and accuracy have been attained.

Having, then, stated the object of this inquiry, before going more deeply into the subject it has been considered advisable to sketch briefly the clinical history of an imaginary typical case of cyclitis; and to follow out its pathological and histological characters in a similar manner, in order that the nature of the process may be more clearly comprehended.

Clinical History of Cyclitis.

Let it be supposed that a chip of metal enters an eye close to the margin of the cornea, and passes backwards through the base of the iris into the vitreous body, in which it becomes lodged.

The eye, if not too seriously damaged by shock, speedily shows signs of the presence of an irritant by its reaction. The conjunctiva becomes injected, and the scleral vessels tortuous. Irritability to light, photophobia, and lacrymation soon became evident. Pain is early present, due, probably, to various causes, such as the movements of the eye and eyelids, irritating the fresh wound, or to the actual presence of a sharp substance in the eyeball. Deep ciliary injection soon appears, even as early as the twenty-fourth hour. The wound has probably closed by this time, but is not firmly healed for many days. The tension of the globe becomes increased, and pain is more marked, especially at night, and is accompanied by a great tenderness of the eye. The pain is markedly circumorbital in character, and is difficult to alleviate. The aqueous humour is turbid.

At a variable time, from the fourth to the eighth day, if the media are clear enough to allow it, a yellowish reflex is seen behind the lens. The anterior chamber, at first shallow, soon becomes deep, and ultimately deeper than normal. The tension of the globe is now stationary, or tends to fall, and will soon become subnormal. The pain at this stage is almost entirely gone.

Pressure on the eyeball, however, brings out the fact that the ciliary region is very tender at some spot, and this, with the presence of engorged vessels of the scleral group, indicates that the eye is in great danger of becoming atrophic.

The tension of the eye continues to diminish slowly, and owing to the pressure of the four rectus muscles, the eyeball comes to assume a somewhat square aspect. This in turn is followed by diminution in the bulk of the eye, and depression of the cicatrix of the original wound.

The eye is now in the condition which is termed "phthisis bulbi."

The whole process has occupied perhaps three months.

Pathological Review of Cyclitis (Figs. 1 and 2).

The course of events, then, in this eye which has led to such complete destruction is somewhat as follows:—owing to the presence of an irritant in the eye, great increase of vascularity has taken place, with formation of an exudate consisting of cell elements in a coagulable fluid. The parts directly injured are united by this exudate. Thus, the iris is probably adherent to the corneal wound, owing to the presence at both the posterior surface of the cornea and the anterior surface of the iris, of an organic substance which, it may be assumed with fair accuracy, is adhesive. The iris and ciliary body become congested, and give rise to an exudation of similar characters to that in the anterior chamber (fig. 1). This exudation soon begins to undergo changes, termed for convenience "formative," and what was a semi-fluid substance becomes a solid, which is, for a period, continually becoming less in bulk (cicatricial contraction), so that the parts to which the exudation is adherent are drawn together. Thus, the exudation which has been formed behind the lens is adherent all round its periphery to the ciliary body, and by its contraction first draws the lens backwards, and then pushes it forwards; but at the same time, being adherent to the ciliary body, it draws this

PLATE VIII.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis ; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 1.—Cyclitis of fourteen days' duration. Radial section through anterior segment of the eye showing, above, cornea to right and sclerotic to left ; mid-distance, the iris and ciliary body ; below, the empty lens capsule to right, and the exudate into the vitreous body to left. The various layers in which the exudate is disposed are seen. × 9 diams.

Fig. 2.—Cyclitis of many years' duration. Showing the corneo-scleral junction above and to the right, part of the displaced and distorted lens to the left. Mid-distance is the displaced ciliary body, and a large mass of fibrous tissue ; and below is the retina separated and crumpled. × 14 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

FIG. 1.

FIG. 2.

Bale and Danielsson, London.

centrally, and separates its posterior part from the sclera, drawing forwards, in doing so, both retina and choroid.

Again, the vitreous body having been permeated by this self-contracting fluid, shrinks towards its points of firmest attachment, namely, the lens and ciliary body, and at last disappears more or less completely. The contraction of the vitreous body is most frequently soon followed by separation of the retina, its support being withdrawn; and it is frequently the case that the retina is firmly adherent to, and involved in, the mass of cicatricial tissue which has slowly accumulated behind the ciliary body and lens (Fig. 2). During this period hæmorrhages from the displaced blood-vessels in the retina and elsewhere may take place, more or less completely masking the appearances.

Lastly, while the above-mentioned changes have been going on, interstitial formation of exudation, and subsequent contraction, causes destruction of the delicate structures and diminution of vascularity, and thus the stage of diminished intra-ocular tension is arrived at, and is closely followed by shrinkage of the globe.

It becomes evident at an early stage in the investigation that there is a very wide degree of difference as regards the date after receipt of an injury of the onset of different stages of the process in various cases.

It may be explained that it was, in the majority of cases, traumatic cyclitis which came under observation, as cyclitis of other origin does not often give cause for enucleation of an eye until a late stage has been reached.

The diversity mentioned above has been attributed to variation in the intensity of the irritant, on the one hand, and of the resistent power of the tissues, on the other. To these same causes was attributed a certain variation in the histological characteristics of the process, noticed early in the investigation. So well marked, indeed, was the difference, that the inflammatory process seen might have been divided into several distinct classes, which

would depend for their distinctions on histological characteristics.

Subsequent investigation of the subject, however, has led to the conclusion that, as many cases partake of the characters of different types, such a differentiation would be likely to lead to confusion rather than to elucidation. Each case seems to have its own characteristics as regards cell elements, fibrous tissue, etc., almost entirely independent of the date of onset of symptoms, few being met with which were quite typical of one particular class.

Hence the subject has been dealt with as a whole, as an average of cases seen, and where deemed necessary special changes have been treated as such, and indication of the stage of the process at which they are likely to occur has been given.

Histological Changes.

Turning now to the investigation of the histological changes seen in such an eye as that under consideration, it is to be noticed that an eye is rarely removed at an earlier date than the eighth day after the receipt of injury, as the loss of such an eye cannot well be certain before this time. Hence the earliest changes can only rarely be seen in the human subject. Of course many eyes are removed within two or three days of the date of injury, but, as a rule, these are so seriously damaged that the reparative process is delayed.

During the course of some work, however, for a different purpose, an opportunity of examining the eyes of animals in an early stage of cyclitis presented itself, and in this way it has been possible to make comparatively satisfactory observation on this subject.

Changes in an early stage of cyclitis.—The earliest recognisable change is, as would be expected, dilatation of the vessels of the uveal tract within the sphere of influence of the irritant, whatever it may be. This may be seen to take place within one hour of the injury. A few hours later multinuclear cells may be seen on the surface of the

ciliary body and the iris, and lying in the angle of the anterior chamber.

The origin of these cells is, doubtless, vascular, their appearance on the surface being the result of opening up of natural channels in the epithelial covering.

The exudation being liberated at all points from what may be considered a free surface into a fluid or semi-fluid medium, much of it is at first carried away by the natural circulation of fluids in the eye, and passing through the pupil goes to form a more or less fluid mass, which accumulates in the anterior chamber as hypopyon.

Hypopyon.—This is found to consist of multinuclear cells —finely granular leucocytes—in a medium which is fluid but coagulable.

The coagulum formed is composed of a dense network of delicate fibrillæ, which join at varying angles, and very frequently have small globules at the points of junction.

In this fibrinous network the cells are caught and detained. The degree of fluidity of the hypopyon seems to depend largely upon the proportion of cells to fibrin, the more cellular exudations being more fluid.

From its situation in the angle of the anterior chamber the hypopyon is quickly removed, so far as can be seen, by breaking down of the fibrinous network, and by either breaking up of the cells or by their escape by passing through the spaces of Fontana into the canal of Schlemm, and thence into the circulation.

The exudate in the posterior chamber, however, is not so readily disposed of, and much of it generally remains to become organised.

Further back along the ciliary body, where the exudate formed passes into the space in which the fibres of the lens ligament lie, the tendency to organisation is even greater, and it is here that the first-formed fibrous tissue is usually seen, even as early as the eighth day of the process.

Pars ciliaris retinæ (Fig. 3).—The cells liberated from the pars ciliaris retinæ pass with the natural current into the vitreous body, along its anterior part towards the posterior

pole of the lens, in a backward direction deeper into the substance of the vitreous itself, or in a backward and outward direction along the anterior part of the retina.

In this way a more or less dense white cloud is formed in the anterior part of the vitreous body, which increases both in size and in density as the process of cell migration advances.

Attention has been called above to the fact that as early as the eighth day it is seen that fibrous tissue is developed in close approximation to the ciliary body, and from the exudation itself. This process of fibrous transformation continues to advance throughout the whole course of the affection and extends in all directions in which the exudate is being formed.

The process of fibrous transformation, which is regarded as of great importance, will be considered later.

Cell Migration from the Ciliary Body.

It has been seen that cellular exudate is found in the vitreous body, then, and it must be shown that these cells do really migrate into the positions in which they are found, that is, to show that they are not produced by proliferation of fixed cells in the vitreous body.

The vitreous body.—The writer's experience of the examination of the histological structure of the vitreous body has been fairly extensive, including, as it has, the vitreous of man at various ages from three months upwards, that of the ox, sheep, pig, dog, cat, frog, and some varieties of birds and fishes. Further, several different means of fixation of the tissues have been used, such as formalin, in fluid and vapour (which is the quicker), osmic and chromic acids, perchloride of mercury, Müller's fluid, alcohol, and lastly freezing (in ice and salt).

The sole means which have been found reliable as demonstrating the existence of any structure in the vitreous body are fixation by alcohol (90 per cent.) and by freezing. The only structure shown by even these last two methods

PLATE IX.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 3.—Cyclitis of twenty days' duration. Section of pars ciliaris retinæ to show the origin and position of the exudate. × 100 diams.

Fig. 4.—Cyclitis of sixteen days' duration. Section has been bleached by euchlorine. To show the origin and position of the exudate, and to compare with Fig. 3. × 100 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer; Fig. 3 excepted, cornea to right.

FIG. 3.

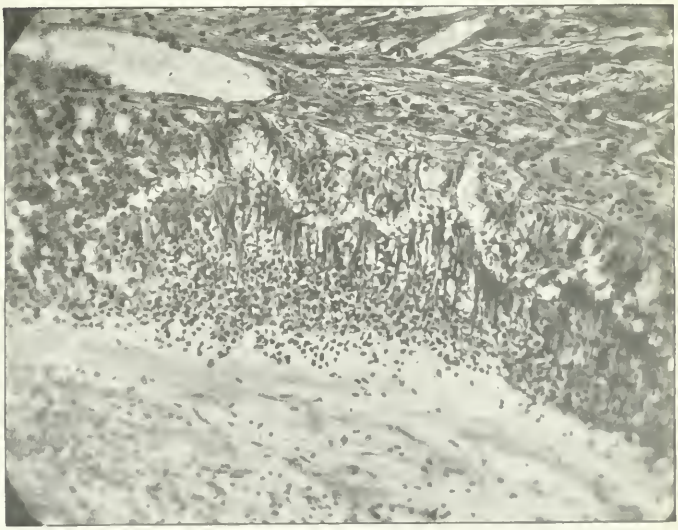

FIG. 4.

Bale and Danielsson

was observed macroscopically. It consisted of indefinite septa, which seemed to pass in a radial direction from the centre towards the anterior and, to a less distinct degree, towards the posterior part of the jelly-like substance. A distinct central canal was never seen, nor has the existence of fixed cells been demonstrated, although staining by different agents in varying strengths and exposures has been resorted to.

The statements regarding the passage of exudation from the ciliary body into the central canal, made by MacGillivray (15), have not been verified, although the subject has been carefully investigated. According to the writer's experience, when the exudation seems to take the form of a cone whose apex is at the optic-nerve entrance, this fact is due to shrinkage of the whole vitreous body to this shape, which, considering the connections of the structure, is a natural one.

Taking it for granted, then, that if there are any fixed cells in the vitreous body they are extremely difficult to demonstrate, it may be assumed that if cells are found in any considerable quantity in this situation they have gained entrance to it from the exterior.

As will be seen later, however, there are in cyclitis not only large numbers of cells found in the vitreous body, but also several varieties of cells. Hence this assumed migration of cells must be accepted as an element in the inflammatory process.

The Origin of the Cells (Figs. 3 and 4).

The next subject for investigation must be the origin of the cells which are found in the vitreous body in cases of cyclitis. It has been shown above that in the very early stage of cyclitis multinuclear cells were found lying, often in considerable numbers, upon the surface of the ciliary body. Similar cells are found in the sulci between the ciliary processes, on the posterior and anterior surfaces of the iris, and in the anterior chamber.

It may be mentioned that the endothelial cells on

Descemet's membrane show no signs of proliferation in, at all events, the early stages of cyclitis.

Local or hæmic origin.—Two views only require to be considered in regard to the origin of the migrating cells: (1) that they are of local origin, derived from the proliferation of fixed cells in any situation near the site of their appearance, and (2) that they are derived from the blood-vessels by migration.

To begin with, the writer was inclined to the opinion that a large proportion of the leucocytes formed were the direct result of the proliferation of the endothelium covering the ciliary body, and more especially of the non-pigmented internal layer of the pars ciliaris retinæ. Further investigation of the subject, however, induced him to modify this view in favour of the hæmic origin of the cells.

The reasons which seemed to point to the local origin of the cells were as follows:—

(1) Whilst, as is seen in a photograph of a bleached section of the ciliary region (Fig. 4), large numbers of cells were accumulated on the surface of the endothelium, and many were seen lying between, and often apparently in the endothelial cells, but few were to be found outside this layer of non-pigmented cells or in the interstices of the pigmented layer.

(2) Whilst the non-pigmented layer was frequently found to be much broken up, it was very rarely found that, except in advanced cases, the pigmented layer was much disturbed.

(3) There was rarely seen any distinct accumulation of cells round or near the blood-vessels in the ciliary body, such as is observed in other situations, as the conjunctiva, the retina, etc., when cells of hæmic origin are migrating from the vessels.

The reasons which, on the other hand, point to the hæmic origin of the cells, and which weighed more heavily than the preceding, were as follows:—

(1) The nuclei of the cells composing the non-pigmented layer were only very rarely seen in a state of active pro-

liferation, although, as mentioned above, leucocytes were found in abundance in the interstices of the layer and close to the nuclei of the endothelial cells.

(2) Large numbers of leucocytes containing pigmented particles were found in the interior of the vitreous, and pigment granules also were found lying free internal to the pigmented layer, indicating apparently that the leucocytes had to some extent broken up the pigmented layer in forcing a passage through it.

(3) The accumulation of leucocytes on the surface of the ciliary body and between the cells of the epithelium might be the result of delay in transference of the cells through the layer of fibrous tissue, which is early formed on the surface of the ciliary body (Fig. 4).

(4) It is seen that, in other situations, where there is a discharge of leucocytes from an epithelial surface, the cells composing the epithelial layer are rarely found in a state of proliferation, even although large numbers of leucocytes may be passing through their interstices to the surface, so that it is quite invalid to assume that those escaping leucocytes are of local origin.

(5) Lastly, whilst it is admitted that the endothelial cells do frequently proliferate and give rise to cells which are multinuclear leucocytes, when such proliferation is going on it is a very manifest change, and can easily be recognised.

In the case, for instance, of the proliferation of the endothelium on Descemet's membrane (as has been so ably demonstrated in recent years by Dr. Reid), the change in the cells is a very manifest one.

Now, as we have seen, this is not so in the case of the ciliary body in the early stages. Hence, it may be concluded that the large majority of the leucocytes found on the surface of the ciliary body are of hæmic origin, and have escaped from the vessels in the ciliary body (which in moderately advanced cases are usually found to contain such leucocytes in large numbers), and have migrated into their new position.

This migration is probably accelerated or facilitated by the existence of perforations in the membrane of Bruch in this situation, as can be demonstrated microscopically (Fig. 4).

Various Kinds of Cells.

It has been seen above that there are in the early stages of the cyclitic exudation multinuclear cells of various origin, but there are also cells which present different appearances. These first appear about the seventh day, and are large mononuclear leucocytes of oval or irregular shape. They are much less numerous than the multinuclear variety. They may be found lying in the spaces, which are usually distended (by œdema) between the layers of the pars ciliaris retinæ, and even outside the pigmented layer, close to the hyaline membrane of Bruch (Fig. 4).

No single origin can be definitely assigned to these cells, as they may be seen in the blood-vessels in the ciliary body, and also in close relationship with fixed cells in the stroma of the ciliary body which are manifestly in a state of active proliferation.

It is probably the case that they are of dual origin, as the fixed cells of the ciliary body do proliferate, even at a comparatively early stage in the inflammatory process.

Whatever their origin, however, they have been closely associated with the change from the fibrinous state to the fibrous condition of the exudation, as will be seen when discussing the individual cell elements.

Fibrosis of the Exudation and its Consequences.

As above noted, fibrous transformation or fibrosis of the exudate begins on the surface of the ciliary body at a comparatively early stage, but it does not interfere, to any great extent at least, with the continuance of the accumulation of cells in the vitreous body.

This fact is due to several considerations, the most important of which are as follows :—

PLATE X.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 5.—Cyclitis of twenty-two days' duration. To show the inner surface of the retina, with the vitreous body separated below. Large epithelioid cells are lying in the space. × 120 diams.

Fig. 6.—Cyclitis of twenty-two days' duration. To show the inner surface of the retina and the adjacent vitreous body. The origin of the exudate in the retina is seen. × 120 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

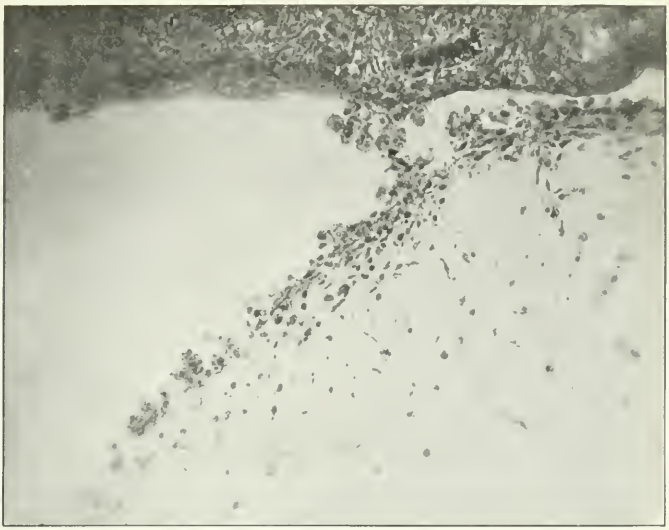

FIG. 5.

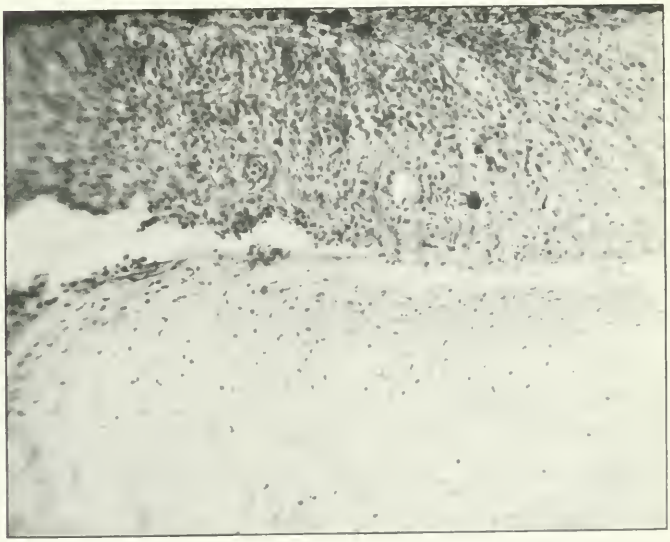

FIG. 6.

Bale and Danielsson, London.

(1) The area of exudation surface tends to increase at a greater pace than does the advance of fibrosis.

(2) The fibrous membrane becomes vascular, and in this way participates in the formation of the exudation.

(3) The cells composing the exudation themselves proliferate, and thus assist in the formation of new exudate.

As these facts are of considerable importance in connection with the histological process which is going on, they may be discussed more fully.

Rapidity of extension of area of exudation (Figs. 5 and 6). —As there is a free surface, so to speak, all round the ciliary body, and as there are currents in the vitreous body which convey in various directions the fluids which enter it, it is evident that, if an irritant capable of diffusion enters the vitreous body, its action will not long remain local. The extensions of the area of irritation and exudation, therefore, depend upon the currents from the ciliary body and in the vitreous body. If, in the early stages, the ciliary body is only in part influenced, the distribution of the irritant will soon set up irritation in other parts, until not only the whole of the ciliary body and iris, but even the anterior part of the retina will participate in the inflammatory process. The endothelial lining on the internal limiting membrane is seen to proliferate, sending off large epithelial cells into the vitreous body or into the space which frequently exists between them (Fig. 5). But the retina itself may be affected, leucocytes may be seen leaving the engorged vessels in the fibrous layer and accumulating round them in groups, and ultimately passing out into the vitreous or into the fluid behind it, if it be shrivelled (Fig. 6).

Thus there is a greatly extended area of exudation, even at a comparatively early stage in the process, and at a period when the process of fibrosis is confined to the surface of the ciliary body and the fibres of the lens ligament.

Formation of blood-vessels in the exudate.—This second consideration is of great importance and interest, in so

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Formation of blood-vessels in the exudate.—This second consideration is of great importance and interest, in so

far as it gives an instance of the powers of new formation of blood-vessels, which seem to follow an exudate through an almost unbroken endothelial membrane.

The means by which this new formation of blood-vessels takes place is not clear. That there are normally minute apertures in the membrane of Bruch at its anterior part, through which in the first instance cells of hæmic and other origin may pass, and in the second through which blood-vessel loops may be protruded from the distended vessels in the vascular layer of the ciliary body, can hardly be denied. Such solutions of continuity can be seen microscopically, and through them it may be that the glands of the ciliary body protrude outwards, as Fig. 4 shows. Capillary loops may, or may not, surround the base of these glands (the writer has not been able to demonstrate this), but, at all events, it is probable that with the demand for an increase of nourishment in the vitreous body, capillary loops developed from the ciliary body pass out at an early stage into the vitreous chamber and take part in the formation of new exudation.

The presence of blood-vessels in the fibrous layer at an early stage, say the tenth day, can easily be demonstrated; but in later stages the vessels are of large size and are present in large numbers, penetrating often to a considerable depth into the vitreous exudation. From these new-formed blood-vessels cells migrate in considerable numbers, thus assisting the formation of the exudate in the vitreous body.

In later stages, as will be seen, the vessels play an important part in the removal of the exudate.

Cell proliferation of the exudate.—This last-mentioned consideration is the activity of the cells which compose the exudate, both fibrinous and fibrous, in dividing, often with what may be considered to be almost violent rapidity, and sending off a progeny which passes forwards and, ultimately, like its parent, will proliferate.

The proliferation of the cells which form the exudate is a very large and important element in the production of

PLATE XI.

Illustrates Dr. Leslie Buchanan's paper on Cystitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 7.—Cyclitis of sixty days' duration. Section to show the process of fibrosis of the exudate. Pars ciliaris retinæ above just visible, stratification of the exudate below. × 200 diams.

Fig. 8.—Cyclitis of eighteen days' duration. The exudate in the vitreous body fibrino-cellular in nature, with a fibrinous band below × 100 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

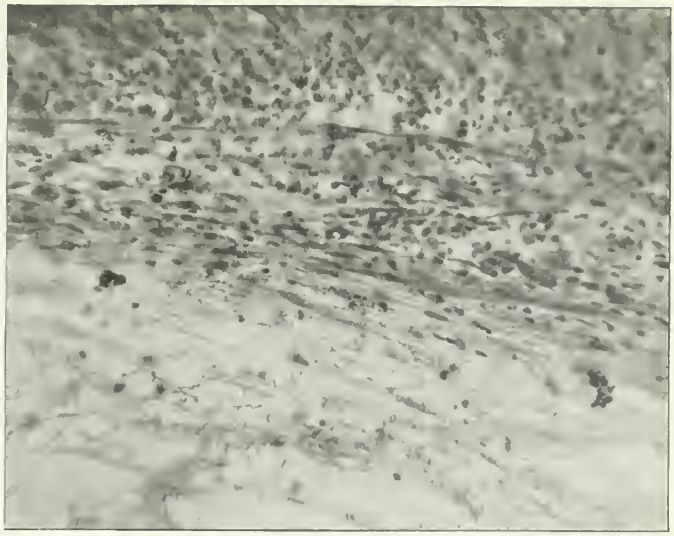

FIG. 7.

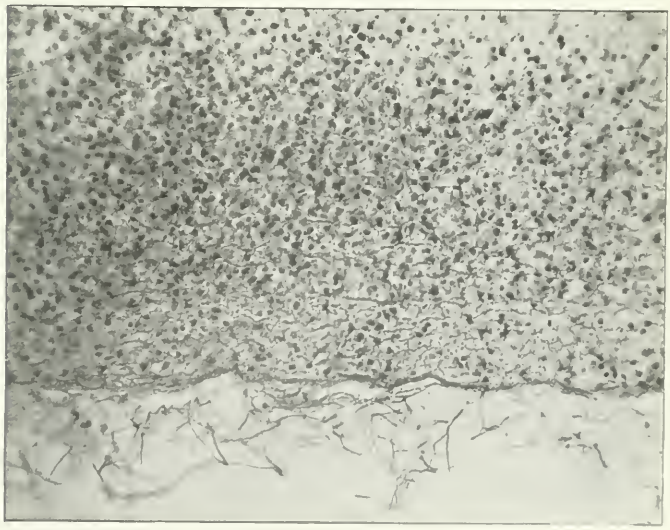

FIG. 8.

Bale and Danielsson, London.

new cell matter, and is by no means confined to one set of cells.

Before going on to describe the means by which this proliferation takes place, however, a few words may be said regarding the condition of the eye when the exudate into the vitreous body is fully developed.

The fully developed Exudate (Figs. 7 and 8).

In the fully formed exudate there is usually a certain arrangement of the cell elements into layers, which is of some importance.

In order to understand this arrangement completely it may be well to enumerate the various structures and layers seen in a radial section through the ciliary body, although Figs. 1, 3, 4, and 7 may give a fair idea of them.

Passing from without inwards (Fig. 1), we find the sclerotic, and to the inner side of it the outer layers of the ciliary body with the ciliary nerves superposed at intervals. The ciliary body is composed of layers of fibrous and muscular tissue, and has on its inner surface a rich vascular layer, which in turn lies closely applied to the membrane of Bruch. To the inner side of this latter lies the pigmented layer of the endothelium of the ciliary body, which is often found spaced out by œdema. Further in one sees the non-pigmented layer of the endothelial covering. These two layers, which compose the pars ciliaris retinæ, are often broken up and pushed apart—as seen in the various photographs, which represent these layers in an early and a late stage of cyclitis,—and occasionally a layer of fibrous tissue is developed between them.

Internal to the non-pigmented layer is found a moderately dense layer of fibrous tissue, which passes by fairly abrupt transition into a thick layer of loose fibrinous exudate (Fig. 7).

Passing more deeply into the vitreous body, and still well defined from the layer of fibrinous exudation, is a

layer of dense cellular exudation, which, as will be seen, consists of cells of various kinds in a fibrinous network (Fig. 8).

It may be understood that the thickness and extent of these layers of exudate in the vitreous body will depend largely upon the duration and intensity of the inflammatory process.

In some cases the masses of deposit, which have somewhat the appearances of clouds, may be of quite limited extent, and may be close to the ciliary body, although not in contact with it. In other cases the whole vitreous body may be occupied by exudation, leaving a translucent layer close to the ciliary body only.

The lens may or may not be infiltrated, according as it has or has not been injured. Even at this stage the cicatricial contraction which has taken place has caused separation of the posterior part of the ciliary body and distortion of the iris and lens.

The Cell Elements in the Exudate.

To return now to the consideration of the arrangement of the cell elements in the exudate, it is found that in the layer of loose fibrinous exudate which lies in an intermediate position, there are very few multinuclear cells in this advanced stage.

In the early stage they were numerous, as above noted, but have now passed more deeply into the vitreous body, leaving in their place, apparently, a comparatively small number of rounded or oval cells of medium size. These are the mononuclear cells noticed before as being seen first about the seventh day, and which have been associated with the process of fibrosis (fibroplasts).

It will be noticed that this fibrinous layer lies close to the fibrous layer, and that it is at its outer margin that fibrosis is advancing.

The process of fibrosis (Fig. 7).—The conversion of

PLATE XII.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 9.—Cyclitis (part of Fig. 8). To show a variety of cell forms in various stages. The dim outline of the "ghost" cells is well marked on account of the position of the part from which the photo is taken. × 300 diams.

Fig. 10.—Cyclitis of twenty days' duration. To show a more cellular part of the exudate than Fig. 9. The outline of the "ghost" cells is more distinct in this position. × 300 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

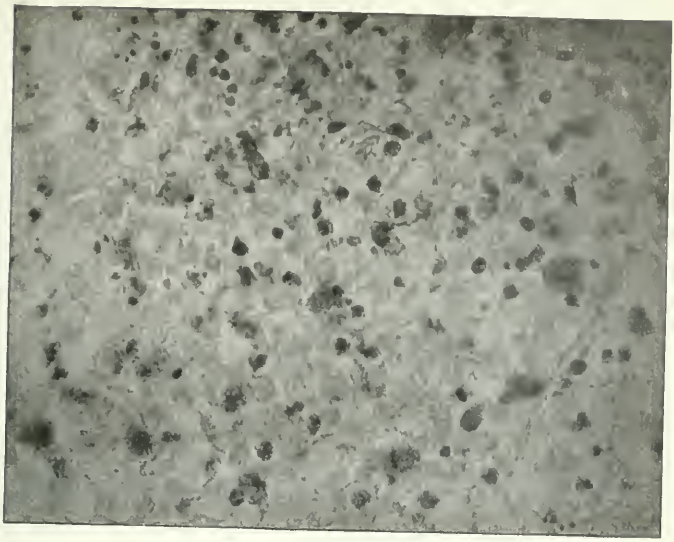

FIG. 9.

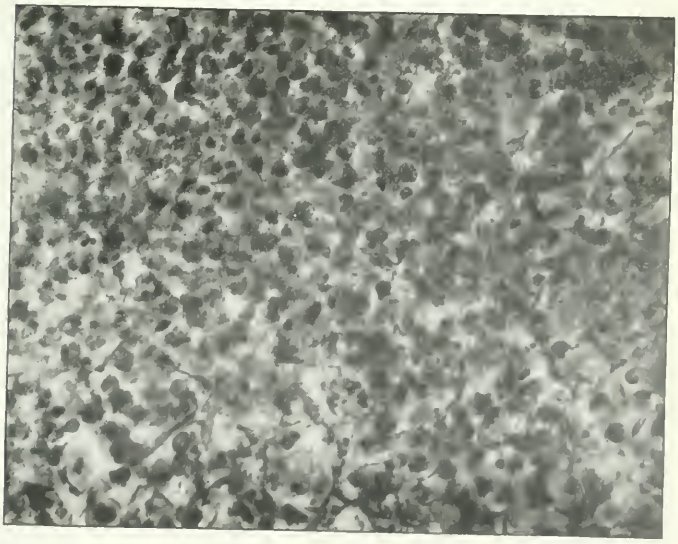

FIG. 10.

Bale and Danielsson, London.

fibrinous into fibrous matter is evidently undertaken from its beginning by mononuclear cells which never penetrate into the exudate further than the fibrinous layer.

The changes are somewhat as follows:—

From being simple, rounded, mononuclear cells the fibroplasts, as they have been termed, become cigar-shaped, or much elongated ovally. The longer axis of the oval is usually found to be parallel with the advancing edge of the fibrous layer. Now it is seen that in the neighbourhood of this elongated cell the fibrinous strands become condensed around the cell itself. A much elongated double cone is thus produced by the enswathing of the cell in fibrin. This is at first opaque and granular, but soon becomes translucent and takes on a form which is indistinguishable from that of an ordinary fibre of fibrous tissue.

The action of the nucleus in this matter is evidently all-important, but whether it has the power of transforming the fibrin into protoplasm—which, considering the appearances, is probable,—or converts the fibrin, by feeding on it, as it were, into protoplasm, cannot be definitely determined.

Cell forms in the exudation (Figs. 8—12).—In the inner part of the fibrinous layer it has been seen that there are but few cell elements. Passing inwards towards the centre of the vitreous, however, a layer is met with in which great cellular activity is going on.

This is a mass of cell matter of varying kinds, in a matrix consisting of fibrinous strands, which form septa passing in various directions and dividing the exudate into loculi (Fig. 8).

The cells are of several varieties, five of which may be mentioned.

(1) A *multinuclear cell* the protoplasm of which is finely granular, and which is actively engaged in proliferation (Figs. 9 and 11).

(2) A *mononuclear cell* of medium size, which is seen to undergo division (Fig. 10).

(3) A *large epithelioid cell*, which is occasionally undergoing division (Fig. 9).

(4) A *small round mass of nuclear matter,* probably a young cell (Figs. 10, 11, and 12).

(5) A *large mass* of more or less ill-defined protoplasm without nucleus (Fig. 12).

These varieties of cells may be accounted for as follows:

(1) The first is a leucocyte which has migrated from a vessel, or is the result of the division of the nucleus of a mononuclear cell.

(2) The second is a lymphocyte, which may be (*a*) of vascular origin, (*b*) derived from the budding of other exudation cells, or (*c*) from the budding of fixed cells in the ciliary body.

(3) The third is derived from the internal surface of the retina.

(4) The fourth is a bud from the proliferation of some other cell in the exudation; it varies greatly in size and can be followed through various stages. This variety is found most abundantly in the most advanced portion of the cellular mass.

(5) The fifth and last cannot properly be termed a cell at all, as it is merely the protoplasm of a cell, the nucleus of which has passed out, either as a whole or in more or less numerous fragments.

Such protoplasmic masses are usually found in large numbers in the less advanced and middle regions of the cellular mass, and are manifestly undergoing degeneration, the less advanced masses being always extremely indistinct, only a vague outline remaining, before they finally disappear in the fibrinous layer. They have been termed "ghost" cells.

Cell Proliferation in the Exudation.

Allusion has been made several times to the subject of proliferation of the exudation cells, and to this subject we now turn, having delayed giving any detailed description of the process in its varying characters until the nature of the mature exudation was explained.

PLATE XIII.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

FIG. 11.—Cyclitis (part of Fig. 9). To show the nature of the exudate more clearly. Evidences of nuclear activity are seen. × 600 diams.

FIG. 12.—Cyclitis of sixteen days' duration. To show "ghost" cells and evidences of nuclear activity in a part of the exudate near the most cellular part. × 650 diams.

NOTE.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

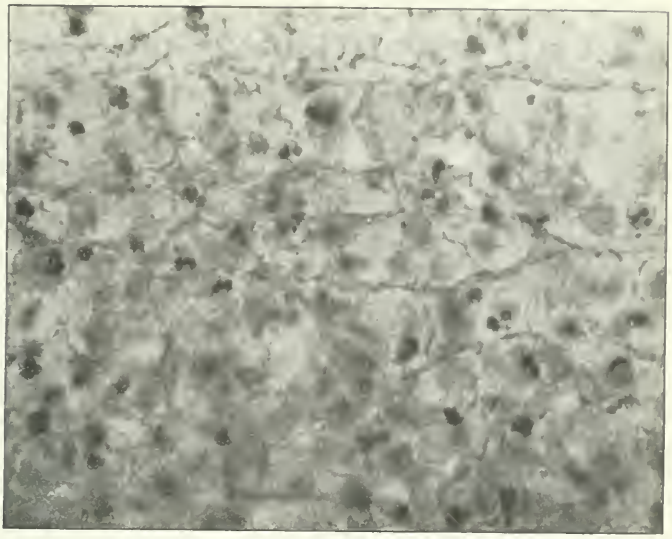

FIG. 11.

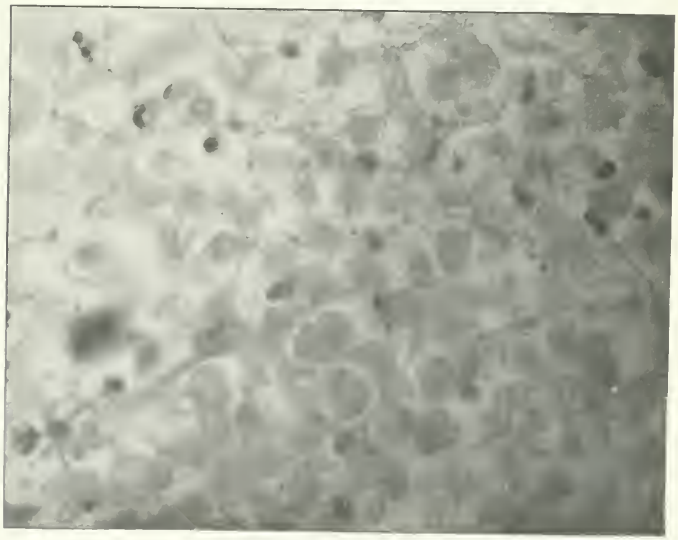

FIG. 12.

Jas. and Pat. Given, Lith.

Four distinct varieties of activity of the nucleus have been observed, and it is to be noted, as specially interesting, that the process termed karyokinesis is not amongst them. They are (*a*) gemmation or budding, (*b*) fission, (*c*) fragmentation of the nucleus, and, lastly, (*d*) total emigration of the nucleus.

Remark will be made later regarding the non-appearance of karyokinesis, and in the meantime a few words will be devoted to each of the methods of proliferation which are so well illustrated in this form of inflammatory exudation.

It is, of course, impossible to observe the actual process of nuclear division, etc., but although the process cannot be followed out in any one cell, the various stages of the change are so frequently seen, even in any one section, that but little imagination is required to enable an observer to arrange them in order and form a series.

(*a*) *Gemmation.*—The first evidences of change are seen in the nucleus, the protoplasm remaining, so far as can be seen, inactive.

First, without any previous manifest alteration in the nucleus, a fine notch appears in some position on the edge of the nucleus. This notch gradually spreads more deeply into the substance, and is seen as a curved line of light, which extends, ultimately, across a small portion of the usually spherical mass.

The appearance is as if a small lid was being lifted off the pole of the nucleus. Later, however, it is manifest that, by extension of the notch, a small spherical portion of nuclear matter is separated from the parent, leaving a cup-shaped depression in this latter. In this cup-shaped depression the daughter-cell lies completely enclosed within the protoplasm of the parent. From this position the new-formed nucleus ultimately departs, passing straight out through the protoplasm, and taking with it, it may be, a small portion of protoplasm to act as a protective envelope. It at once comes within the sphere of influence of the irritant, and passes outwards and onwards, building up

for itself as it goes from the nutrient fluid surrounding it, a thicker protoplasmic layer.

This new-formed cell at first has the appearance of a small lymphocyte, but soon gains bulk and, ultimately, like its predecessor, if necessary gives off a progeny which is capable of repeating the process above described.

The mother-cell can, apparently, give off a large number of buds; indeed a nucleus can give off, as has been seen, more than one bud at a time.

(*b*) *Fission.*—The parent or primary cell may divide by simple division of the nucleus into equal parts, each portion taking a share of the protoplasm with it.

(*c*) *Fragmentation of the nucleus.*—In this process the whole of the nucleus is divided into small masses, of spherical shape, with great rapidity. Each of the small portions passes out from the protoplasm, leaving what is virtually an empty husk. Their departure is probably almost simultaneous, as many of these little nuclear masses may be seen lying outside a parent protoplasm husk in the fibrinous network.

(*d*) *Migration of the nucleus.*—Lastly, the nucleus of a cell may take a worm-like shape and project itself into a new space entirely, or may, apparently, project itself partially out of its protoplasm, and in this new position a small portion may be cut off and remain as a separate nucleus, the parent retracting into its protoplasm.

In the former instance an empty husk is left, as in the last-described method. It is hardly necessary to remark that the empty protoplasmic husks left are the "ghost" cells.

The appearances presented by these masses of empty cell protoplasm being many and differing somewhat widely, gave rise to considerable conjecture when they were seen at first; but subsequent study of the subject has established the correctness of the views taken. Considering the vast numbers of these bodies seen, they must remain visible for a considerable time after being left by the nucleus, and before being disintegrated, as they eventu-

ally are. Further, they are always more fresh or natural-looking near the advancing edge of the proliferating zone, and more faded or "ghost-like" near the border of the fibrinous layer of exudate.

This subject forms matter for much interesting speculation, indicating, as it does, that the protoplasm of a cell is a purely passive agent, and also that the position of maximum activity in an exudation is constantly changing. The latter statement is supported by the fact that in the most advanced edge of the exudation zone it is almost entirely new-formed cell matter which is found.

From the foregoing it will be evident, then, that cell proliferation in the exudation is very active, and that it is a highly important part of the function of an inflammatory process. It provides that in the front rank of what may be compared, not inaptly, to an army advancing into a hostile country, young and active units are constantly present; and, further, it ensures that rapidity and, probably, endurance will be present when they are most urgently required. But as the urgency of the case in point, in many, if not in all, instances depends upon the proximity and virulence of micro-organisms in the tissues, it may be well to turn aside to the investigation of the presence of organisms in the exudation.

Micro-organisms in the Exudation.

It is not difficult to demonstrate the presence of organisms in the exudation, and, by comparison of sections, to ascertain the position which they occupy. Thus they have usually been formed in the more advanced portions of the exudate. It is usually micrococci, staphylo- or streptococci which are found, but occasionally a somewhat doubtful bacillus has been seen. The bacteriological investigation of the subject has not, however, been closely followed out, as the information to be derived from the knowledge of their presence is solely connected with their influence on the exudation.

From the facts that organisms are usually found only in

the advanced edge of the exudation and in the interior of cells, it may be concluded that the cell activity is intimately connected with the removal of the organisms. Further, it is only rarely that the presence of organisms can be shown in the ciliary body or choroid, and then, also, it is in the interior of cells that they are found.

Function of the Exudation.

These various appearances have led to the adoption of certain views regarding the function of the exudation in this inflammatory process, as being those which best satisfy the requirements of the case :—

(1) The migration of cells from one position to another is not a passive but an active response to a definite demand which is termed chemiotaxis.

(2) This chemiotaxis is in response to a very well-defined stimulus for a purpose which is probably dual. In the first instance active proliferation is required, and in the second active phagocytosis.

(3) It is highly probable that the mature cell is not able to move with the same rapidity as the young cell, and also the mature cell has not the same power of resistance as the young cell.

(4) As living protoplasm is to be pushed forward into the neighbourhood of, and indeed, into close proximity and contact with noxious matters, be they animal, vegetable, or chemical, it is necessary that this protoplasm should possess the maximum activity and resistive power when first brought into such contact.

(5) The cells of the exudate having these functions, the fluid and semi-solid matrix acts as a support and a nourishing medium.

Before going further, the absence of evidences of karyokinesis may be inquired into.

Karyokinesis not Seen in the Exudate.

It is suggested that the absence of this type of cell

proliferation may be readily explained if it be accepted that it is a less rapid method of division than the others.

The writer has had abundant opportunities of examining inflammatory exudates, as formed in pneumonia, pleuritis, and corneitis, and although he has investigated, in the case of the cornea especially, many sections in regard to this subject during the past four years, no instance of undoubted or even doubtful karyokinesis has come under notice in an acute inflammatory process. Only once, indeed, has the process been seen in the true corneal tissue, and that in a case of very slow form of inflammatory change.

All the various modes of division may be seen in the cornea (acutely inflamed), and several varieties in other situations in which a similar change is advancing. Again, in the epithelium on the anterior surface of the cornea in the ordinary state karyokinesis is frequently seen. In the case, however, of rapidly growing epithelium, which is extending over a cut surface as a protective agent, as occurs within a very few minutes, no instance of karyokinesis was seen. Here, then, is an instance in which cells do show karyokinesis under ordinary circumstances, but when required to reproduce with great rapidity, do not manifest it.

With these various facts in support may it not be contended that the process of karyokinesis is not sufficiently rapid for the exigencies of the case?

Leaving this part of the inquiry, we will turn to the investigation of the means by which the exudate is removed and a further supply stopped.

The Cessation of Exudation.

As would be expected, the process of exudation will stop not suddenly, but more or less gradually, and the cessation will depend upon the removal of the cause. In this way, when all the irritant (and its source) has been removed, or enclosed in a protective envelope, the demand for new cells will cease. The vessels will then begin to

contract, and diapedesis will be lessened and ultimately checked. But the proliferation of the exudate itself will not be stopped so abruptly, and many new-formed cells endowed with the power of rapid proliferation will be formed. The chemiotactic attraction will be wanting, however, and consequently activity will be no longer required, and no longer shown. There will still, however, be a considerable bulk of cell matter lying unoccupied, as it were, and the process of removal of this must now be investigated.

The Absorption of the Exudation.

It has been seen that the areas over which the exudate is formed, and over which it is spread, are both large, and also that, even in an early stage in the inflammatory process, change is going on in the exudate, which results in its assuming a more stable form. As it is largely with this same change or fibrosis of the exudate that we are now concerned, it is important that the positions in which the exudate is found should be again enumerated.

Sites in which the Exudation is Deposited (Figs. 1 and 2).

First, the posterior surface of the ciliary body from the iris to the retina is covered with a dense mass of more or less completely formed fibrous tissue.

Backwards from the ciliary body a mass of exudate, likewise undergoing fibrosis, is found to pass round the posterior surface of the lens. Extruding further backwards still the exudate is found to reach a greater or less distance, presumably according to the intensity of the irritant. From the ciliary body again, backwards and outwards along the surface of the retina, a considerable bulk of the fibro-plastic mass may extend even to the equator of the eye.

The position in which the largest mass of tissue is

PLATE XIV.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis.

Fig. 13.—Cyclitis of two and a half months' duration. To show a late stage of the exudate undergoing fibrosis and fatty degeneration. × 100 diams.

Fig. 14.—Cyclitis, seventeen years after. To show the formation of true bone in the curve of the displaced ciliary body. × 15 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

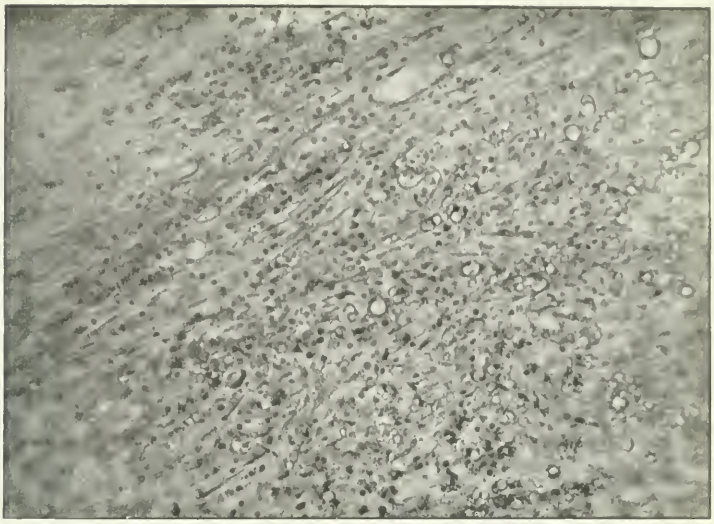

FIG. 13.

FIG. 14.

accumulated is just behind the ciliary body, where, owing to cicatricial contraction, the ora serrata has been drawn forwards, and the mass of exudate lying over it is brought up to increase the bulk.

In close proximity to the fixed tissues, however, fibrosis has been going on, but in the central parts of the vitreous body the exudate is entirely fibrino-cellular. It is with increasing difficulty, as can be understood, that nourishment is conveyed to this central area, at this stage. Much of the vitreous body has been destroyed, and the remainder is useless as a circulating medium. Moreover, it is from the ciliary body that nourishment is to come, and, as is seen, this region is cut off to a large extent by fibrous tissue, which is now contracting on itself, occluding the vessels which formerly acted as a source of supply. Hence it is seen that many of the cells of the vitreous exudate undergo fatty degeneration, as seen in Fig. 13. The protoplasmic matter degenerates and the nuclei, which have probably undergone proliferation, are left to assist in the process of cicatrisation by fibrosis. Fibrosis advances rapidly all round the mass of exudate, and large distended blood-vessels may be seen, but the circulation is sluggish in them. Fatty degeneration of the tissue goes on, and it is many weeks, even months, before the mass comes to a state of rest. All fatty *débris* must be removed, all superfluous cells taken away, until ultimately the fibrous tissue is hard and firm like cartilage, possessing little or no vascular supply.

Much variety in detail is observed, however, by one who examines many cases of cyclitis in later stages.

Thus, if the amount of cellular matter in the vitreous body is large, cicatrisation goes on all round it, and ultimately a solid mass of spherical shape, white color, and great firmness, remains in the centre of the much contracted vitreous body.

Again, if the vitreous body has been almost entirely replaced by exudation, it is frequently the case that this, containing as it probably does, much noxious matter which

is being removed, cannot be got rid of in such a simple manner, but sets up a secondary irritation which causes erosion of the coats of the eye, and in this way, by perforation of the globe, finds an exit as pus.

In extreme cases, where the irritation has from the first been of very high intensity, the inflammatory process spreads to all the tissues of the eyeball, and by the exudate degeneration forms pus, which can only be removed by perforation of the sclerotic or opening up of the original wound. Such a case is termed "panophthalmitis."

In all cases of cyclitis, then, it is seen that a certain amount of fibrous tissue may be left to contract to a quiescent state, after the inflammatory symptons have passed away, and in this remaining fibrous tissue another change may take place, namely, ossification.

Ossification of the Exudation (Figs. 14 to 18).

This change has been recognised for nearly a century now, and consists in the formation of true bone in the exudate. This is ossification as distinct from calcification. In both cases there is a deposition of lime salts in the tissues, but whereas, in the former, such salts are acted upon by living cells, in the latter they remain inert and passive in an amorphous or crystalline mass.

Both these forms of deposition are found more or less commonly in the eye after inflammatory changes, but the one is common in situations where the other is rare. In the cornea, for instance, lime salts are frequently deposited, and may assume the crystalline or amorphous form, but bone, the result of organic influence on the salts, does not form.

In the situations where bone is most frequently developed, simple calcification is not found. In the lens, however, we have a common meeting ground where calcification is very common and bone is occasionally developed.

Calcification is a mere mechanical deposition of lime

PLATE XV.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 15.—Cyclitis, eleven years after. To show an early stage in the formation of bone in the exudate. The lower and more advanced portion bears some resemblance to bone; the less advanced portion is opaque and granular. × 15 diams.

Fig. 16.—Cyclitis (same as Fig. 15). To show the advancing edge with its calcareous particles.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

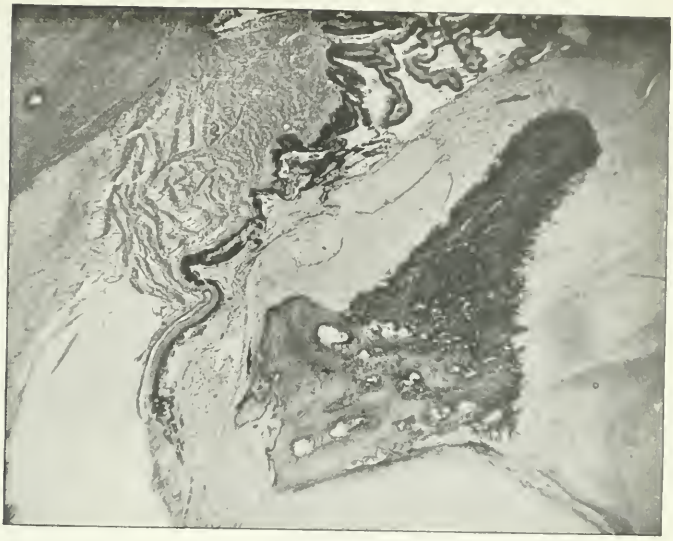

Fig. 15.

Fig. 16.

Bale and Danielsson, London.

salts, but bone is a transformation of the tissue which is formed after inflammatory changes have subsided.

The formation of bone in the eye is to be regarded as entirely distinct from the formation of bony plates in the sclerotic, as it occurs in certain fishes and reptiles, and is not an ossification of a cartilaginous plate, such as is found in the sclera of some birds. Nor is it to be compared with the formation of plates of bone in tendons, etc., which are in direct continuity with the periosteum. Finally, it is to be distinguished from the ossification of secondary growths of tumours which are growing in connection with bones.

The process here dealt with is practically an ossification of a cicatrix, and, so far as can be ascertained, the formation of true bone in a cicatrix elsewhere (unconnected with periosteum) is unknown.

Two cases are known in which bone has been developed in a cicatrix which has replaced blood-clot after an extensive bruise to a limb, one of which may be quoted here.

On January 13th, 1899, Mr. F. S. Eve showed to the Clinical Society of London a case in which a large plate of bone was developed apparently in the substance of the vastus externus muscle, and was separated from the femur by a distinct interval. It was the sequel to the formation of an extensive blood-clot in the thigh, following a kick from a horse. Mr. Eve expressed the opinion that this was an instance of ossification of fibrous tissue derived from the organisation of a blood-clot.

The second case has not been published, so far as can be found, but was in many respects similar to that quoted.

Now in this interesting case there is a possible source of osteoblasts, in that the periosteum might have been ruptured, so that a single or multiple centre of ossification might have been the starting point of the osseous change.

In the case of ossification of the fibrous tissue deposit in the eye, however, there is no question of direct connection with bone or periosteum, yet some definite cause must exist to explain the comparative frequency of the occurrence.

A possible explanation is suggested in the facts that a direct prolongation of the brain is exposed to danger of injury, both by contraction of the fibrous tissue in the eye, and by external agency. Further, as far as is possible, all the great nerve centres are preserved from such injury in bony cavities. Hence it may be the case that in the instance under consideration, the means of protection being within easy reach, and still, until converted, a source of danger, this fibrous tissue is converted into bone for a similar reason. Again, the fact that the fibrous tissue developed in the eye is lying in close proximity to a highly vascular membrane, may have some influence in bringing about the change of conditions. No matter what may be the actual cause of the transformation, it is exceedingly difficult to explain the determining cause, or in other words by what path osteoblasts are conveyed to the eye; for, even admitting that calcification of cicatrices is not uncommon, the conversion of this mineral matter into bone is only accomplished by the intermediation of specialised cells, which are uniformly present in such cases.

In the case under discussion, then, it must be admitted that the process of formation of bone is undertaken in answer to a definite demand, and that the necessary osteoblasts are either brought to the eye from some other situation by the circulation or are formed locally.

As regards the actual formation of bone the writer has come to the conclusion that the process is somewhat as follows.

The formation of the bone (Figs. 15 and 16).—In close proximity to a blood-vessel, and probably from it, minute granules of spherical shape appear at some point in the mass of fibrous tissue. These granules gradually increase in both size and number. Those which were first formed coalesce, whilst new ones remain for a time separate. These globules show by their reaction to acids that they are composed of lime carbonate, and by their coalescence form amass of what seems at first to be an inorganic lime salt (Fig. 16). The mass thus formed is homogeneous

PLATE XVI.

Illustrates Dr. Leslie Buchanan's paper on Cyclitis; a Study of the Inflammatory Exudations into the Vitreous Body in Cases of Cyclitis (p. 208).

Fig. 17.—Cyclitis, after thirteen years. To show the structure of the fully-formed bone. A cavity lined with osteoblasts is seen. × 150 diams.

Fig. 18.—Cyclitis, after twenty years. To show the connections of the growing bone with the surrounding structure. Fibres are spread out, and the cells are arranged between them. × 150 diams.

Note.—The plates are arranged so that the whole eye would lie with the cornea away from the observer.

FIG. 17.

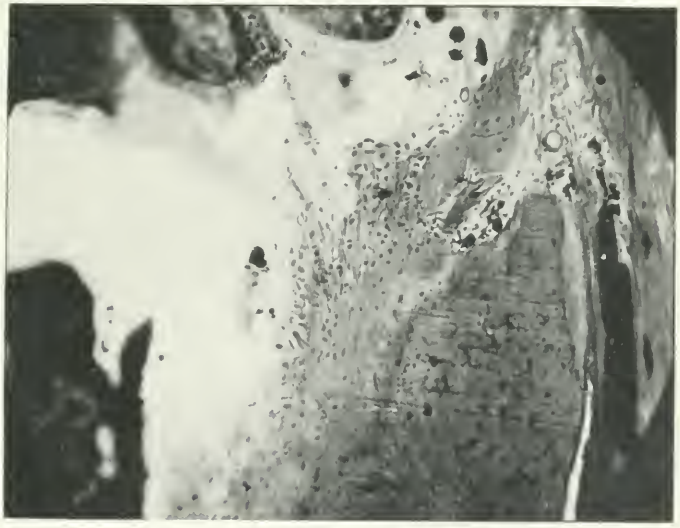

FIG. 18.

Bale and Danielsson, London.

in appearance, of considerable density, and stains with great readiness. Its edges are ragged and terminate in globules, which diminish in size as they are further from the mass.

At an early stage, long delicate fibres, and occasionally stellate corpuscles, may be seen lying between the globules and radiating from the mass into the surrounding tissue. These are evidently comparable to the osteogenetic fibres and osteoblasts. The more advanced portion of the mass thus formed early encloses the blood-vessel, from which it may be said to have originated, and soon shows a marked change in structure. Formerly dense and homogeneous, it soon exhibits a fine striation, as if indicating an arrangement in layers round the somewhat irregular cavity in which the blood-vessel lies. This cavity becomes more open and its limits are well defined.

At a somewhat later stage it is seen that the spaces are lined with osteoblasts, which are regularly arranged, and in the lumen of the tube, as such spaces really are, besides the blood-vessel, fat, and often fatty crystals in sheaves are found. In the completely formed bone the cavities tend to open up even more, and the striation which was seen before becomes better marked. At this stage it can be clearly seen that there is a definite arrangement of the bone corpuscles along the margins of the layers, and of the whole structure around the blood-vessels (Fig. 17).

The *growing end of the bone* continues to be ill-defined, and consists usually of a series of fibres, which pass outwards into the surrounding fibrous tissue. Round these fibres it is apparent that the cells which are about to enter into the formation of the bone arrange themselves in columns; and as the process advances it seems as if the fibres and cells are both surrounded by the calcareous deposit (Fig. 18).

It is of interest to notice this fact regarding the cells of the fibrous tissue, as such a conversion into bone corpuscles surely indicates a certain power of adaptation as regards functional activity.

We have thus seen that there is formed in connection with, and probably by conversion of, the fibrous tissue of the cicatrix in the eye, true bone, and it only remains to be mentioned that this ossification is by no means rare; indeed, the comparative frequency of occurrence of ossification in the eye is in a certain class of cases remarkable.

During six years' work as pathologist in the Glasgow Eye Infirmary, the writer has examined over 600 eyes, and has found that bone was developed in over fifty of them. Now a large proportion of these eyes examined (600) were removed in early stages of cyclitis, or on account of other diseases in which no fibrous tissue is developed in the eye, so that, if one takes only those eyes which have been rendered liable to the conversion of fibrous tissue into bone, the percentage will be found to be not far from fifty-five or sixty. As regards the age of the patients in whose eyes bone has been developed, it is interesting to note that, although for various reasons the average is over forty, many quite young persons of either sex are so affected. The youngest patient whose eye in the writer's experience has contained bone, was aged twelve, whilst the oldest was seventy-seven years. The shortest period which has elapsed between the date of injury and the discovery of bone was eight years, and the piece of bone found was of almost microscopic dimensions—1 mm. long and ·50 mm. thick, only. It will thus be seen that the formation of bone in the cicatrices in the eye is a comparatively common occurrence, that it occurs in both old and young subjects and in both sexes. Now, as before remarked, the formation of bone in cicatrices in other situations in the body, if it does occur at all, is of extreme rarity; may we not then argue that there is a special reason in case of the eye for its development.*

Having thus briefly followed out the main features of the process termed cyclitis, and having dwelt at some little

* The writer does not consider that the formation of bone in an eye increases the danger of sympathetic ophthalmitis.

length upon points which appeared to be of special interest, the writer is bound to acknowledge that the subject is by no means exhausted. Much could have been added, and the whole subject might have been gone into in fuller detail.

Literature.

It is to be regretted that there appears to be no literature dealing as a whole with the subject chosen for investigation. It is true that a moderate amount of work has been done from an ophthalmological point of view, including clinical observations upon cyclitis with a few notes upon the principal features of the exudation, and a considerable bulk of literature upon "ossification of the eye," as it has been termed. The subject of this investigation, however, has hardly been dealt with in detail in any work, and thus it has been more than usually difficult for the writer to obtain assistance by comparisons of his results with those of others.

The best recent text-books on ophthalmology give but a cursory glance at the histological processes involved. Thus, Fuchs (1) says that "the exudate consists of fibrinous strands with cells in varying proportions," whilst the subject is similarly briefly dealt with by the writers of the article on the subject in Norris and Oliver's (2) work on ophthalmology. Weichselbaum (3) refers to the subject briefly, and gives an illustration in many ways similar to one of the writer's photographs (Fig. 5).

Indeed, the various works, histological, pathological, and ophthalmological, to which an observer would turn for assistance, seem to show that no detailed research has been made in this subject.

The first point which has been found to be dealt with in looking over the literature of the subject is the origin of the leucocytes which are found on the surface of the ciliary body. Thus, Haensell (4) says that proliferation of the

cylinder cells of the pars ciliaris retinæ takes place by indirect division. Again Alt (5) says, "more frequently we see that these layers (the uveal and retinal layers of the ciliary body) take an active part in the formation of the ciliary membrane, especially the retinal part of the ciliary body," and "the cylinder cells of this layer proliferate and begin to grow out into long spindle-cells, and finally into long connective tissue fibres." This, then, is apparently in line with the opinion at first inclined to, but subsequently rejected, in favour of the hæmic origin of the cells. The latter part of Alt's view could not, however, be accepted at all.

Regarding the occurrence in the exudation of tubules, pigmented or non-pigmented, considerable prominence has been given to it by various writers.

The writer has only once seen such tubules (non-pigmented), and was satisfied that they were, in this case at all events, the result, as Collins (6) has suggested, of proliferations and hyperplasia of the glands associated with his name, and situated in the pars ciliaris retinæ.

At all events such structures are by no means essential, but are rather accidental in the formation of the ciliary exudate. Structures resembling tubules, the result of oblique sections being made of the ciliary processes, are, of course, often seen.

Hamilton (7) remarks upon the occurrence of proliferation of the endothelium of the peritoneum in inflammatory conditions of that membrane.

The subject of the early transformation of fibrinous into fibrous tissue is of interest, as indicating, possibly, an attempt to protect the ciliary body, as well as the fact that all necessary conditions are present at an early stage.

Turning next to the subject which is regarded as of most interest and importance, namely, the proliferation of the leucocytes which compose the exudate, it is seen that it has not been very fully investigated. For many years it has been contended for and against, and the balance has been gradually turning in favour of the view that the cells

which compose inflammatory exudates do actually proliferate. Hamilton (7) thinks that it is probable that many of the cells composing the exudate in croupous pneumonia may be of such origin. Hohnfeldt (8) remarks in his work on abscess formation, that fragmentation of the nuclei was observed in the central area, but that karyokinesis was not seen either in the leucocytes or the connective-tissue corpuscles. Several writers regard this process of fragmentation of nuclei as an evidence of degeneration, but it might as well be taken to be a sign of great stimulation of a living cell. The protoplasm, when left by the nucleus, may, and does degenerate, but the process of fragmentation is seen in the class of case under consideration so frequently associated closely with evidences of great vital activity, that the weight of evidence is rather against the assumption that the nuclei are degenerating.

Regarding other evidences of proliferation of cells in the exudation, indication of such great activity in inflammatory conditions of the cornea is so abundant that, here as there, there is no doubt but that the process is one of high importance in the production of the exudate and the maintenance of its activity.

The absence of karyokinesis has been commented on before, and it is only to be noted here that it has been contended that many cases of supposed direct division have really proved to be indirect. The precautions which have been taken in the preparation of tissues and sections, however, render it somewhat improbable that the true nature of the process has been overlooked.

The presence in the exudation of large numbers of rounded masses of protoplasm without nuclei ("ghost" cells) is of interest. No reference to the occurrence of similar structures in the exudate and other situations has been found. This may be attributed to various facts. The condition of transparency, as found in the vitreous, is absent elsewhere; degeneration of such bodies and subsequent removal may be more rapid elsewhere, owing to differences in vascularity of parts. The staining affinities of the bodies

are indifferent, which fact renders them, in either recent or old condition, liable to oversight, and also, it may be added, difficult to photograph. It is imagined that, even indistinctly as they are shown in the photographs, the various considerations of size, relations, position, and appearance in various states being taken into account, it will be difficult to account for the presence of such bodies in any manner more satisfactory than that given.

The evidences of motility of the nucleus are also of interest, and are of frequent occurrence in the exudate, especially in the less advanced portions.

Generally speaking, it may be concluded from the appearances seen, that the activity of the leucocyte is by no means restricted after its passage into a new sphere, but rather increased, possibly by the intervention of new conditions and the presence of an irritant.

Further, it is clear that the *rôle* of a leucocyte in the exudate is a many-sided one, including phagocytosis, production or proliferation, and repair, so far as it is possible, of the tissues. Adami (9) concludes from various appearances that "the new growth to replace the tissue destroyed by the irritant proceeds *in the main* from the fixed cells of the tissue."

Regarding the process of ossification of the fibrous exudation, the opinion has been expressed that the osteoblasts may be derived from specialisation of the connective-tissue corpuscles.

Twelve years ago, unknown to the writer, this view had been taken by Dr. Reid (10), who says, "The connective-tissue corpuscle seems to be transformed into the bone corpuscle."

Although much has been written on the subject of ossification and many cases recorded, the early stages (or what seem to be such) of the process have been but little investigated. This may be accounted for by the fact that centres of ossification are naturally difficult to find. Although bone may be present in the eye in masses varying in size from a microscopic point to complete conversion of the contents of the sclerotic—as in one case which the writer

has seen,—unless it is found by accident, when it occurs in small particles, its presence is not sought.

The first writer who appears to have seen the true relationship of the process in its pathological aspect was Mackenzie (11), who pointed out that bone was usually found in the eye only after the occurrence of long-continued inflammation. Hoene (12) remarks that an embryonal tissue is laid down and undergoes fibrous changes, after which bone is deposited. Had this subject been more fully grasped when the occurrence or non-occurrence of ossification of the lens, iris, retina, and vitreous was being discussed by Knapp (13), Alt, and others, the possibility of such changes would have been seen at once. Alt (5) records a case of bony deposit in the lens cavity within the crumpled lens capsule; De Wecker (14), ossification of the retina; and the writer has seen bone formed within the limits of the iris, lens, and retina. Regarding the actual process of bone formation, it is admitted that the method described may not be the only one possible. Alt thought that ossification of the choroid, or, as he said, near the inner surface of the choroid, very frequently originated from the vitreous bodies (verrucose excrescences) on the lamina vitrea.

In conclusion, as regards the whole subject dealt with here, it may be the case that, although there is an apparent diversity of appearance between the changes seen in the exudation into the vitreous body in cyclitis, and those seen in inflammatory exudates elsewhere, the diversity is more apparent than real. The nature of the tissue into which the exudate is thrown must have a very powerful influence upon the amount of detail seen. Thus in the case of pleuritic exudate the resemblance is strong, whilst in pneumonia it is not so strong. In keratitis, on the other hand, there is in many of the varieties seen no apparent resemblance at all, only close investigation revealing the fact that the fibrinous matrix is the chief element of difference, the cells behaving in much the same manner in the various processes. So, indeed, it may be, and pro-

bably is, the case that different forms of the inflammatory process, catarrhal, fibrinous, and purulent, are only different in degree, the changes involved being much alike.

Histological Methods employed.

Hardening and fixing have been carried out by the use of chromic and acetic acids, as in Flemming's solution, without osmic acid, and by formalin in 5 per cent. solution. The formalin gives the best results in the preservation of the vitreous body and its exudate.

Bleaching has been accomplished by the use of euchlorine solution, made by adding to 1 drachm of chlorate of potassium 3 drachms of strong hydrochloric acid, shaking in a stout stoppered bottle, and then adding 3 oz. of water. Sections, after fixing in any medium, are soaked in the fluid for twenty-four hours, and then washed for six hours in running water.

Decalcifying has been carried out in the chromic acid solution, with 10 per cent. of hydrochloric acid added.

Various stains and mounting agents have been used, according to the requirements of the case.

Sections have been cut in gum, except when bone was being examined, when celloidin was preferred on account of the liability of the bone to fall out if gum is used. A Cathcart's microtome was used in the first, and a Jung's in the second case.

Photography has been carried out by the use of Zeiss's apochromatic lenses and projection eye-pieces.

Most of the sections photographed have been stained with Ehrlich's hæmatoxylin solution.

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(4) Haensell.—Recherches sur la Cyclitis, *Bull. Clin. Nat. d'Ophtalm. de l'Hosp. de Quinze-Vingts*, 1884, ii, 49.

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(15) MacGillivray.—The Hyaloid Canal and its Relation to Cyclitic Exudation, *Scott. Med. and Surg. Journ.*, 1898 (October). (*June 13th*, 1901.)

The President said he was sure he voiced the feelings of every one present when he cordially thanked Dr. Leslie Buchanan for his contribution. It was difficult to know which to admire most, the excellent slides or the lucid explanations. It was a paper which would form a valuable contribution to ophthalmology.

XV. MISCELLANEOUS.

1. *Voluntary bilateral nystagmus.*

By H. Work Dodd.

Mr. R. S—, aged 23 years, an organist, can produce at will rapid conjugate lateral movements—nystagmus—of his eyes. His vision and refraction are practically normal. He says, "When I desire to produce the movement, I fix my eyes on some object in front of me and *simply wish* to cause the vibration." When desired to show the phenomenon, he fixes his head, looks forward, opens his lids wider, and the nystagmus appears. There is no great effort involved in the procedure, although rarely it has made him feel dizzy and inclined to have a headache. He can perform while looking up, down, or laterally, at a distant object or at a near one; also while under the influence of a mydriatic. He says the things looked at move from right to left, and *vice versâ*, and thus only does he become aware of the vibrations. He has never been able to "shake his eyes" in any other directions. There has never been any indication of involuntary movements. He states that he can perform best when well and vigorous in health, when standing, and when looking straight in front at a distance. He believes that the "movements are caused by the muscles of the eyes working independently of each other, on the right and left sides of the eyes, like a pianist performing a shake between two notes with his fingers."

He possesses a remarkable control over his ocular muscles, being able to converge vigorously, etc., when desired.

He first found out that he possessed this peculiar

accomplishment about fourteen years ago, by accident, when staring hard at another boy in school.

There is apparently nothing indicating the existence of nervous disease in his present state, nor in his family history.

He has a patch of old choroido-retinal trouble in the upper and outer area of the right fundus, showing an absolute scotoma, which, however, does not affect his direct vision. This dates only from eight years ago, and therefore is quite independent of the nystagmus.

(*Card specimen. October 18th,* 1900.)

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2. *The orthoptic treatment of convergent squint in young children.*

By C. Worth.

Consider first the treatment most generally adopted in cases of convergent squint, and its results. After retinoscopy under atropine, glasses are ordered to be worn constantly. In about 30 per cent. of constant monolateral squints the optic axes, after a time, resume their normal relative directions, and maintain them so long as glasses are worn. These children are usually considered "cured." The other 70 per cent. are left without further treatment, except perhaps the occasional (and doubtful) wearing of a pad over the sound eye, until the age of seven or eight years, when most of them are operated upon. Take first the 30 per cent. in which spectacle treatment alone caused disappearance of the deformity. A certain proportion of these acquire binocular vision and are really cured. The majority, however, continue to suppress the retinal image in the eye which formerly squinted, even though some of these have good vision in each eye. Next consider the 70 per cent. which are not "cured" by spectacles alone. These have usually to wait until the age of seven or eight years before anything else is done. In the meantime the

deviating eye becomes blinder and perhaps its power of central fixation is lost. The eyes may then be put "straight" by operation. But only a small proportion, even of those cases which have good vision in each eye, will ever acquire any sort of binocular vision, even although most persevering orthoptic treatment be then carried out. The time has gone by when a strong "desire for binocular vision" might easily have been established in suitable cases. For, whereas the power of central fixation and the sight of each eye separately are innate functions, binocular vision is an acquired art, and must be learned, at least in part, in infancy or early childhood.

The reason why the treatment of convergent squint has been so extremely unsatisfactory is that the cause of the malady has not been understood. A discussion of the ætiology of squint would be outside the scope of this paper. But I may say that convergent squint is not a muscular affection, neither is it primarily due to hypermetropia, nor to anisometropia, nor to congenital amblyopia, nor to fright, nor to "fits." *The one essential cause of squint is a defective development of the fusion faculty.* In the presence of this fundamental cause any of the subsidiary influences just mentioned may be the immediate excitant of the deviation; in its absence they are powerless to cause squint.

Binocular vision is the psychical act of blending the two sets of impressions received from the two eyes. The fusion faculty is the potential faculty of binocular vision. Thus a child with a high degree of convergent squint may, during the continuance of the deviation, have his fusion faculty trained and developed by the means which I am about to describe. While he is looking into the instrument which I show you, and also after the cure of the deviation, this trained fusion faculty manifests itself in the act of binocular vision. In investigating the fusion faculty in any considerable number of patients one finds that those who see binocularly naturally arrange themselves into three separate and distinct groups, according to the degree in

which they possess this faculty. These grades of binocular vision may be called—

1st grade.—Simultaneous macular perception.

2nd grade.—True fusion with some amplitude.

3rd grade.—The sense of perspective.

The results of orthoptic training of squinters seem to show that the fusion faculty normally reaches its full development before the end of the sixth year of life. Training of the fusion faculty at a time when this should normally be developing is, in suitable cases, exceedingly easy and rapid, and charming in its results. Orthoptic training deferred till a later period is very tedious and disappointing—in fact, from the patient's point of view it is seldom worth the trouble.

I do not purpose to discuss the whole subject of the orthoptic treatment of squint. I wish merely to describe, as briefly as possible, the means by which I have endeavoured to train the fusion faculty at the earliest possible age. I say nothing, therefore, of the many excellent stereoscopes, fusion-tubes, etc., nor of Cuignet's plan of "controlled reading," because these can only be used for older people, and when the angle of the squint is small, and when the suppression of the vision of the squinting eye is not very profound or has previously been overcome by other means.

There are three great difficulties in the way of orthoptic treatment of squint in young children:

1st. The child will only submit to the treatment so long as he finds it attractive and interesting.

2nd. The suppression of the vision of the deviating eye.

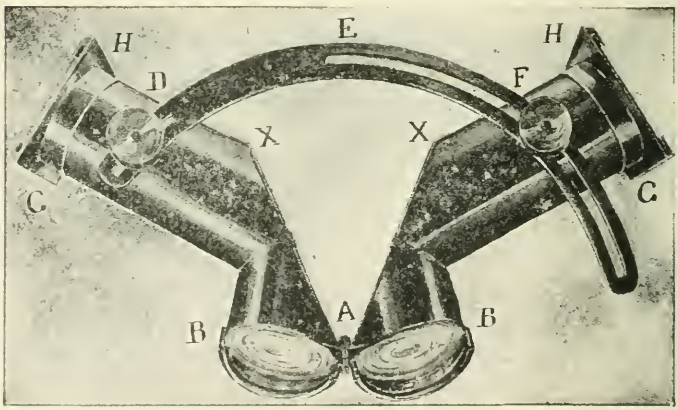
3rd. The angle of the squint is usually so great that, even if there were no suppression, both eyes could not look, at the same time, into an ordinary stereoscope.

In 1895 I made an instrument,* in tin, by means of

* The instruments are made by Mr. Hawes, 74, Leadenhall Street, London, E.C. Now that a considerable number are in use, it is desirable that the instrument should have a special name. I have called it an "Amblyoscope," an instrument by means of which an amblyopic eye is trained to take its share in vision.

which I have endeavoured to get over these difficulties. I have the honour of showing it you to-night, together with a brass copy of the original. The instrument consists of two halves joined by a hinge at A (see Fig. 17). Each half consists of a very short tube joined to a longer tube at an angle of 120°. At A X is an oval mirror. At G H in each tube is placed a small translucent glass object slide. At A B is a lens whose focal length equals the distance of the reflected image of G H. In addition to these lenses there are slots at A B into which other lenses, from the trial case, may be put to correct the refractive error of the

Fig. 17.



The amblyoscope.

patient. D E F is a brass arc with two slots in it—a short slot in which is the binding screw D, and a long slot in which is the screw F. When the screw F is loosened the two halves of the instrument can be brought together to suit a convergence of the visual axes up to 60°, or separated to suit a divergence of as much as 30°. When the screw F is tightened and D is loosened an amplitude of movement of about 10° only is permitted.

Each tube is lighted separately by an electric lamp placed in the direction of its axis. A brass pillar about

Fig. 18.

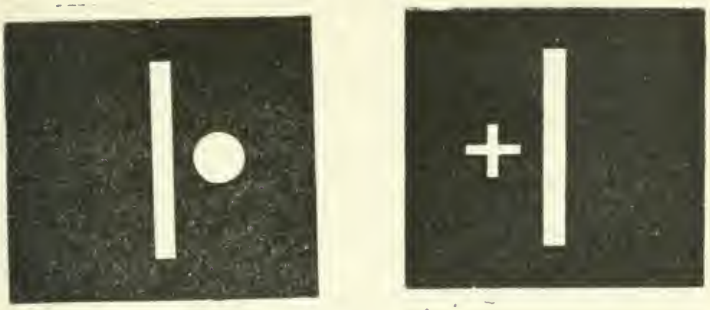


Fig. 19.

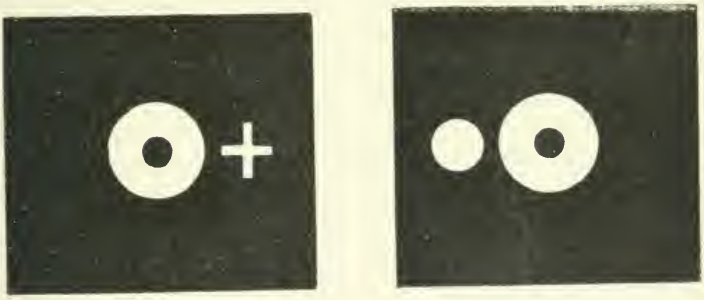
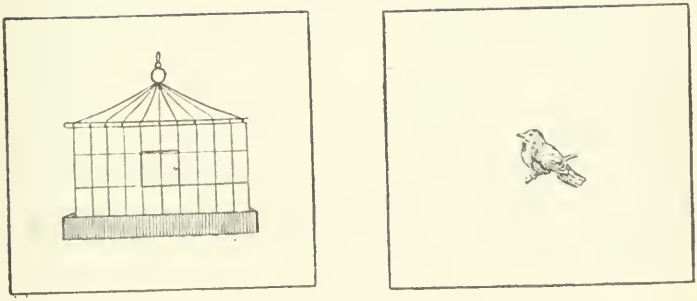


Fig. 20.



four feet high (such as is used to support a reading desk) is placed on the floor three or four feet from the patient. Two light arms, each four feet long, extend horizontally from the top of the brass pillar. Each arm revolves freely in a horizontal plane. An electric lamp is suspended under each arm from a ring which slides along it. The illumination of each of the tubes may thus be increased or diminished by bringing its lamp nearer or pushing it further away. Before I had electric light I used two paraffin lamps on a table, varying the distance of each lamp and the height of the flame as required, a simple plan that does quite as well.

As regards the object slides, each slide consists of a device drawn on translucent paper and gummed on to a piece of glass one inch and a half square.

Figs. 18 and 19 are the familiar round discs and vertical slits with their control marks. These are, of course, for older patients, as one cannot expect young children to take more than a brief glance at such uninteresting objects.

Fig. 20, the bird and the cage, is a type of another set of devices. These do not require any blending of images, but only simultaneous vision. Another set, of which Figs. 21 and 22 are examples, require true blending of the images in order that the full picture may be seen.

Pictures such as Fig. 23 can only be appreciated by patients having the third grade of binocular vision—the sense of perspective. In making the slides, I draw, on pieces of paper one inch and a half square, rough simple designs, such as a clown, a horse, a cow, a man with a hat and pipe, etc., the more grotesque the better. I then make two tracings of each on translucent paper, omitting a different part of the picture in each. To save trouble Mr. Hawes has had blocks made for printing some of these slides. I also have some tiny transparent stereoscopic views for the sake of variety. But "stereoscopic views" are of no practical use whatever in the treatment of squint.

I find that, if one is to undertake the training of the fusion faculty with a fair prospect of success, the visual

Fig. 21.

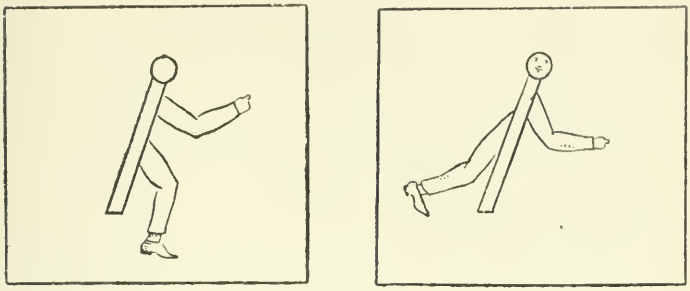


Fig. 22.

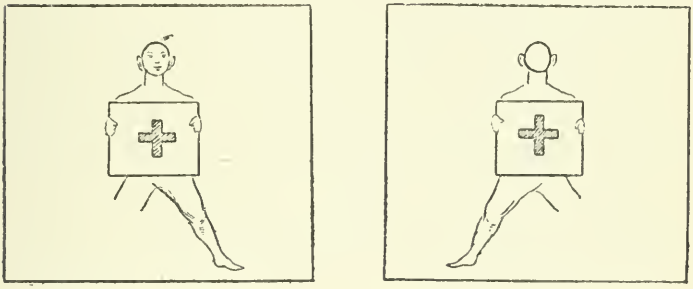
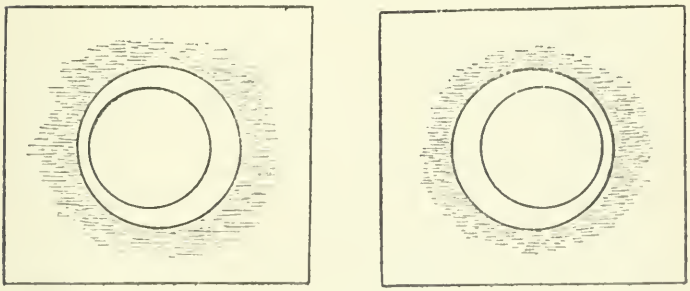


Fig. 23.



acuity of the deviating eye should be not much less than one fourth that of the fixing eye.

Ivory ball test.—In order to estimate approximately the visual acuity in young children I use five little ivory balls, varying from half an inch to one and a half inches in diameter. The child is first shown the balls with both eyes open. Then one eye is covered by a pad, or, if he wears glasses, by a piece of cotton wool stuffed behind the spectacle lens. He is then asked to go and pick up the balls as they are thrown on the floor to a distance of five or six yards, one by one, beginning with the largest. By spinning the ball in the fingers as it is thrown, it can be made to "break" on touching the floor, so that it does not go quite in the direction in which it appeared to have been thrown. It is easy to tell, by the way in which the child runs for the ball, whether he really sees it before he starts or is only going to look for it. I test the presumably better eye first, so as to give the other eye the benefit of experience. Children are always ready to play this ball game, but sometimes a child objects to have the better eye covered. In this case I order the better eye to be covered for an hour a day for a few days, after which there is scarcely ever any trouble. This method of vision-testing takes only a few minutes, and it succeeds with most children who are old enough to walk. I have used it since 1896, and in cases in which I have subsequently been able to test the vision by means of Snellen's types I have found my conclusions confirmed.

The treatment.—Take as an example a child of about four years of age, with a monolateral convergent squint, and who is able to see the one-inch ball with the deviating eye at about six yards. The child either wears his glasses, or lenses correcting his refractive error are placed in the slots provided in the instrument for this purpose. I take the child on my knee and adapt the amblyoscope* roughly

* I used to drop the instrument and break some part of it about two or three times a year, until Dr. Ernest Maddox suggested the simple expedient of slinging it from the ceiling by means of a string and pulley and little leaden counterpoise. Since then there have been no mishaps.

to the angle of his squint. The light which illuminates each tube is, at first, about four feet away. The cage (Fig. 20) is put in the tube before the fixing eye, and the bird in that before the deviating eye. The child at first sees only the cage. He is now told to look out for the bird, while the light before the fixing eye is pushed further away and that before the squinting eye is brought nearer. As the illumination of the object slide before the deviating eye becomes more intense, a point is at length reached when the vision of this eye can no longer be suppressed, so that the child sees the bird he was told to look for. He will now have lost sight of the cage before the fixing eye. The relative intensities of the light are then re-adjusted, and, after a short period of alternation of the vision of the two eyes, the child sees both the bird and the cage simultaneously. The angle of convergence of the instrument is then varied before the child's eyes till he sees the bird enter the cage. The screw F in the long slot is then tightened and the screw D in the short slot is loosened, so that a movement of about 10° only is permitted. Many other similar pairs of slides are now shown to the child, who is usually very curious to see what the combined picture will be, so that if at first he sees only one object he will look diligently for the other. He is now shown a pair of slides such as Fig. 21 or 22. He at first sees each imperfect picture separately (*e. g.* one man *minus* an arm, the other with a leg missing). In a favourable case he very quickly learns to fuse them into a complete picture. Several similar pairs of devices are then shown till he becomes quite proficient in fusing them. He has now (under these special conditions of light and convergence) the first grade of binocular vision—simultaneous macular perception. All this may, as a rule, be accomplished easily in one lesson.

The patient does not easily lose what he has gained in these exercises. At the next visit he will at once begin again where he left off.

The next step is the acquisition of the second grade of

binocular vision, *viz.*, true fusion with some amplitude. This is rather more difficult, as there is a wide gap between grades one and two. The intensities of the illuminations and the angle of convergence of the instrument are arranged as at the last visit. Many pairs of devices similar to Figs. 21 and 22 are successively shown to the patient, and he is encouraged by one's remarks to examine every part of the fused picture. After a time it is found that the angle of convergence of the amblyoscope may be varied very slightly without the fused picture coming to pieces, showing that a "desire" for fusion has been acquired.

The second grade of binocular vision having thus been learned, there is never any great difficulty about the third grade—the sense of perspective; in fact, a patient in acquiring the second grade usually gets the third grade also. The slides (Fig. 23) are shown, and the child is asked whether he is looking at the inside or the outside of the tub. He will at once say "the inside." If these slides are now changed from one tube to the other he will see the tub bottom up. I know no better test than this for the "sense of perspective."

The patient having now acquired the highest grade of binocular vision, under these special conditions of light and convergence, the next step is to equalise the light before the two eyes. This may now be done without a return of the suppression.

After a little practice, with equal illuminations, it will soon be found that the amblyoscope may be converged through a considerable angle, and to a certain extent diverged also, while fusion is maintained. The "amplitude of fusion" may be taken for practical purposes as a measure of the extent to which the fusion faculty has been developed. It must be remembered that the object of these exercises is the training of the fusion faculty at an age at which this may be successfully accomplished—not a mere remedying of the deviation. In many cases, however, the strong "desire" for fusion thus established

directly brings about a cure of the squint. In other cases a visit once a month is quite sufficient to preserve the fusion faculty, while optical treatment is being tried. If the periodic measurements of the angle of the deviation show that this is not decreasing reasonably quickly, and if I have been able to develop the fusion faculty, I have no hesitation in operating at any age, by advancement of an external rectus with or without tenotomy of the rectus internus. The eye being put approximately "straight," the "desire" for binocular vision fills up any slight gap that may remain, and a perfect cure results. If I have not been able to develop the fusion faculty I do not usually operate before eight or nine years of age.

When the fusion sense has been successfully trained during the continuance of a squint, patients do not as a rule experience any diplopia, but one not infrequently meets with cases in which occasional diplopia is complained of, especially when the angle of the deviation is small. I have often heard the report that these children have said they see "two mothers" or "two nurses."

The special points of the amblyoscope are—(1) it can be used during the continuance of a squint of any degree; (2) the suppression of the vision of the deviating eye is quickly overcome by unequal illumination of the object slides; (3) the character and variety of the devices interest the child, so that it can be used at a very early age with a patient of average intelligence, a point of the greatest importance. More can be done in three or four lessons at an age when the fusion faculty should normally be developing than by years of patient work when the child is old enough to undertake voluntarily a course of exercises.

So far only monolateral squints have been considered. Some alternating squints do not essentially differ from monolateral squints. There is usually a high degree of hypermetropia, and the squint alternates, probably because the visual and refractive conditions are about the same in each eye. They are amenable to treatment. The majority

of alternating squints, however, are of a different nature. They begin usually at a very early age, are associated with little or no hypermetropia, and the patients never acquire binocular vision. I have notes of a great many of these cases in which I made very persistent attempts at training the fusion sense, but without success. While looking into the amblyoscope they use the eyes in rapid succession, but the fusion sense is quite absent. These cases would appear to be examples of a reversion to a primitive type, in which the fields of vision were quite independent.

The age at which glasses may first be ordered with success depends to a certain extent upon the disposition of the child, but chiefly upon the degree of the refractive error. A child whose hypermetropia and astigmatism combined amounts to three or four D. will usually wear glasses as soon as he is able to crawl. I have successfully prescribed glasses to a great many children under twelve months of age.

Occlusion of the fixing eye by a pad or bandage has been employed, especially by some French oculists, with these objects:—(1) to overcome the suppression of the vision of the deviating eye; (2) to improve the acuity of vision of the deviating eye; (3) to preserve or restore central fixation in the deviating eye.

(1) Some surgeons order continuous occlusion of the better eye for very long periods, with a view to overcoming the suppression of the function of the deviating eye. I do not use this treatment, because, as already explained, I attain the same object much more quickly and by far less severe means.

(2) The fixing eye is sometimes ordered to be covered with the object of exercising the squinting eye and so improving its visual acuity. I have seldom seen any marked good from it in children who are old enough to read, but have often recorded a slight improvement.

(3) The regular use of the shade every day will prevent deterioration of vision of the deviating eye or loss of its power of central fixation. When the central fixation has

been recently lost and the eye wanders without taking up any position of false fixation, continuous occlusion of the better eye for a week or two will sometimes restore central fixation in a young child. But when the eye takes up a definite position of false fixation the case is quite incurable.

In the case of an infant with a monolateral squint, it is useless to tell the parents that the fixing eye must be covered for so many hours a day for two or three years. Whatever they may promise at the time, they will soon find the amount of supervision required too great a tax on their time and patience. Instead of using the shade, I order one drop of liq. atropinæ to be put every morning in the *fixing* eye *only*. The child will still use the atropised fixing eye for distant vision, but unless the deviating eye is extremely amblyopic (V. $< \frac{6}{60}$) he will look at near objects with this deviating eye, in which the power of accommodation is not abolished. If he does not immediately acquire the habit, occlusion of the fixing eye for a day or two will teach him it. I use the drop of atropine every morning in the *fixing* eye *only*, for the first fortnight in each month. I have used this plan for several years, and I have never seen central fixation lost when it has been adopted; and since I have used the ivory ball test I have not noted any deterioration of visual acuity of the squinting eye.

(*October 18th*, 1900.)

Mr. Hartridge said he had carried out such exercises in his private practice for two years or more, but in hospital work there was not time to do so, at all events by any elaborate plan. The method he had adopted was, after careful optical correction, to cover up one eye for a certain time each day by means of a little metal clip to slide over the glass. Patients were apt to dodge this, so he preferred to put on a pad of cotton wool behind the spectacle glass; that applied to very young children. In older children he tried to educate them by means of the stereoscope, and he had invariably found it a very severe test for binocular vision. He had always thought that bar-reading was a very

great help with older children, though it was not an absolute test. It required much practice and perseverance, on the part of both surgeon and parent, to carry out that treatment thoroughly. From the explanation he had heard, it seemed that Mr. Worth's instrument was more complicated than Mr. Priestley Smith's "Heteroscope."

3. *The determination of the influence of the eyeball on the growth of the orbit, by experimental enucleation of one eye in young animals.*

By W. Ernest Thomson, M.D.

(With Plates XVII and XVIII.)

When visiting Moorfields in 1898, I observed that in suitable cases Mules's operation was preferred to enucleation. I had heard and read that enucleation in the young subject resulted in faulty development of the orbit, and it occurred to me that such introduction of a glass globe into the sclerotic ought to minimise failure of growth.

But on making inquiries I found that opinion was divided among surgeons as to whether such failure of development really occurred or not. There seemed, however, to be a feeling that although often stated, it was probably mythical.

There appeared to be little doubt that the subject was one which would be most profitably attacked from the experimental standpoint.

Shortly afterwards there appeared the Report of the Committee of this Society on the relative value of excision and the operations which have been substituted for it. This report * enumerated as one of the disadvantages which have been alleged against simple excision, "the faulty development of the orbit and face on the side from which the eye has been removed when the operation has been performed in

* *Trans. Ophthalmological Society*, vol. xviii (1898), page 256.

early life." The report goes on to say, "Though often referred to in ophthalmic literature, we have been unable to find any definite evidence in support of this suggestion." Then follows the statement that Mr. Gordon Byers (at that time Junior House Surgeon at Moorfields) had carefully measured, in adult life, the orbits in ten cases in which the eyeball was removed in childhood, and had found no appreciable arrest of development, at any rate of the external orifices of the bony walls of the orbit.

Further, in answer to a letter which I addressed to a well-known member of this Society, with reference to my proposed experiments, he replied as follows:—"I fancied the growth of the orbit question after removal of an eye early in human life had been practically settled, *viz.*, that supposing no artificial eye be used, the growth of the conjunctival and palpebral space would be more or less arrested, but the bones would do as they would if the eye were present."

Mr. Treacher Collins, who was a member of the Committee mentioned above, informed me that he knew of no experimental work done to settle this point, and remarked that although the Committee had been unable to find any evidence of faulty development of the orbital margins, there might still be failure of growth in parts of the orbit inaccessible to clinical examination.

Unable myself to find any literature on the subject of experimental enucleation, and convinced that it was quite reasonable to suppose that the eyeball should influence the growth of the orbit, I set about determining the point by experiments upon animals.

The animals chosen were rabbits, because they are the easiest to obtain and to maintain in the limited accommodation at my disposal. It would have been impossible to keep half a dozen dogs or cats for several months, although admittedly they would have been better for the purpose. Even the rabbits were most uncomfortably quarrelsome.

It was rather late in the year before work could be commenced, and there was considerable difficulty in obtaining the animals young enough. Eventually eleven rabbits were obtained, none of which were probably much over twenty days old. Their eyes were open, and they were just able to nibble, and to maintain themselves.

One eye in each of these animals was removed under ether. Antiseptic treatment was carried out. The orbits were plugged with wool soaked in perchloride of mercury solution.

Of the eleven rabbits six survived. It is worth noting that the six survivors were of the common grey wild species, while the five which died came of a domesticated stock, and were of varied colours. The operations were carried on between the 6th and 20th September, 1898.

The six survivors maintained a rapid growth and remained in perfect health during the time they were kept alive. They were eventually killed with chloroform, one in March, at the age of six months, two in April, and three in June, 1899.

It is to be regretted that I did not make an attempt to photograph them during life, as the asymmetry was exceedingly striking. Indeed, it early became obvious that the result would be a positive one, for the animals presented a markedly lop-sided appearance, which seemed to be largely due to loss of prominence of the supra-orbital ridge. After death, when the remaining eye was excised, it became clear that the whole orbital border was contracted on the anophthalmic side. During life several of the orbits were noticed to be occupied by a tumour-like growth, and this was afterwards found to present a modified glandular structure, presumably representing lacrymal gland.

Of the six heads, three were macerated for me by Mr. Eggleton, of the Kelvingrove Museum staff. The other three were kept in preserving fluids, and one was sawn through lengthwise.

It was originally my intention, in conjunction with my

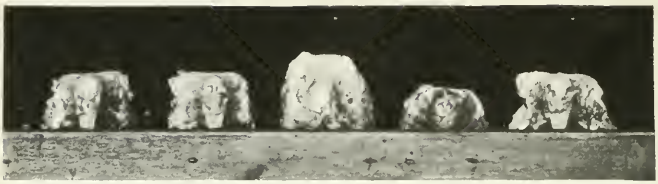


Fig. 1.

Fig. 2

Fig. 3.

Fig. 4.

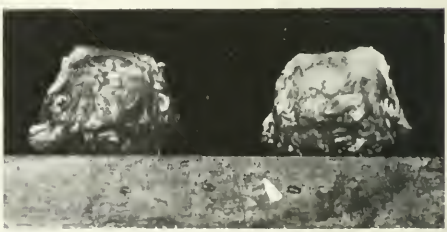


Fig. 5.

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PLATE XVII.

Illustrates Dr. Ernest Thomson's paper on The Determination of the Influence of the Eyeball on the Growth of the Orbit, by Experimental Enucleation of one Eye in Young Animals (p. 258).

Fig. 1.—Five heads with the soft parts (skin excepted). Seen from before. The central head has the lower jaw attached. All the heads show deformity.

Fig. 2.—Two heads with soft parts, normal side. One head has lower jaw attached.

Fig. 3.—The same, anophthalmic side. The difference between the orbits in this and Fig. 2 is well marked.

Fig. 4.—Two heads with soft parts. Seen from before.

Fig. 5.—The same; from behind. In this and the previous figure the droop of the supra-orbital ridge is most marked.

PLATE XVIII.

Illustrates Dr. Ernest Thomson's paper on The Determination of the Influence of the Eyeball on the Growth of the Orbit by Experimental Enucleation of one Eye in Young Animals (p. 258).

Fig. 6.—Dry skull. Seen from above. This photograph very truly represents the deformity of the frontal bone. Notice the extreme deformity of the supra-orbital portion, and especially of the post-orbital process.

Fig. 7.—Dry skull. Seen from below. The left is the anophthalmic side. The supra-orbital ridge is visible on this side, because of its drooping. The photograph, however, in this case exaggerates: on the normal side some of the supra-orbital ridge can be seen from below, although the photograph fails to show it.

Fig. 8.—Dry skull. Seen from before.

Fig. 9.—Dry skull. Seen from behind. Notice in this and previous figure the great alteration in the position of the supra-orbital ridge.

Fig. 10.—Dry skull, normal side.

Fig. 11.—Dry skull, anophthalmic side. Great care was taken to photograph these two sides from the same point of view. Notice that practically every portion of the anophthalmic orbit is deformed.

Note.—Figures 6—11 are all of the same skull.

Fig. 6.

Fig. 7.

Fig. 8.

Fig. 9.

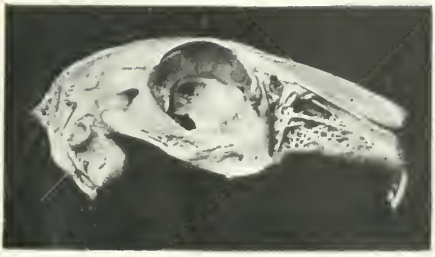

Fig. 10.

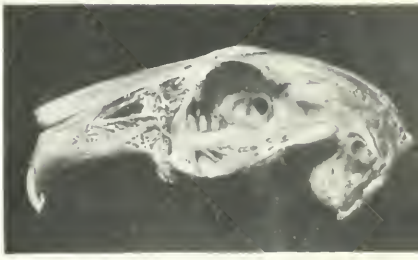

Fig. 11.

Bale and Danielsson Ltd

friend Dr. Leslie Buchanan, who has helped me greatly at the operations and otherwise, to have removed the brains from these skulls in order to trace the atrophied fibres. But so much damage was done to the skull in the only case in which the experiment was tried, that this idea was abandoned.

I will now show you the skulls, both wet and dry, and also the skulls of two control * rabbits about the age at which these others were operated on (see Plates).

The anophthalmic orbit is smaller than its fellow in nearly all, if not in quite all, directions. I have made measurements (in millimetres) of both the wet and dry skulls as carefully as I could, but, of course, such measurements cannot be looked upon as absolutely accurate. Some of them are not even attempts at absolute measurement, but these are, relatively speaking, fairly correct as regards the comparison of the two sides. These measurements have all been put into tabular form (see Tables I, II, and III).

It was especially difficult to estimate the depth of the orbit, because the lower orbital margin on the anophthalmic side seems to be slightly nearer the middle line than that of the normal side (Fig. 7), and cannot therefore be taken as an accurate fixed point relatively to the orbital depth. You will, however, probably agree with me that, apart from actual measurements, the curve of the orbital floor is encroached upon all round, forming as it were the surface of a smaller sphere than that of the normal side (Figs. 10 and 11).

Regarding the tabulated figures as a whole, one is struck by the frequent occurrence of the figure 2 and a fraction as expressing the difference between the chief normal and anophthalmic measurements of the bones in the dry state.

Look at Table II, where the three heads 3, 4, and 5 have been measured *after maceration*. In the antero-posterior direction the differences between the two sides are 3, 1·2, and 2·8, average 2·3. In the vertical, 2·9, 2·2, and 2·9,

* The control skulls are not included amongst the plates.

average 2·6. From the top of the orbit to the last molar tooth, 3·7, 2·3, and 2·3, average 2·7. In three other columns there are averages of 2·5, 2·3, and 2·3.

I do not intend to spend time over the figures in detail, but to deal rather with the averages. Full particulars will be found in the tables of measurement. The orbital margins were measured in their greatest antero-posterior and vertical dimensions.* What I may call control measurements were from the top of the orbital margin to the last molar tooth, and from the junction of the zygomatic process and malar bone to the bottom of the supra-orbital notch and post-orbital fissure respectively. The letter N stands for normal, A for anophthalmic, and D for difference between the normal and anophthalmic sides.

The mean of the average differences between the direct antero-posterior and vertical measurements of the two sides is 2·495, and in the case of the control measurements 2·585.†

In order to ascertain if the lower or zygomatic-malar portions were affected, the distance was taken from a point in the middle line of the head between the orbits to the last molar tooth on each side. The average difference here is only ·70 for three skulls. I think this is what one would expect, for the lower portion of the orbit will be largely influenced by the growth of the teeth and muscles of mastication, especially of the temporal muscle, which in the rabbit occupies a considerable portion of the combined orbito-temporal fossa.

It seems clear that the length and height of the bony margins of the orbit are almost uniformly diminished, and that facial deformity has resulted.

Regarding the depth of the orbit, apart altogether from the bony ridges forming its margins, I have found, as before mentioned, no satisfactory way of getting fixed points.

* The antero-posterior measurement is from the orbital angle anteriorly to the tubercle, giving attachment to a slip of the temporal muscle posteriorly. The vertical measurement is the greatest inside measurement.

† It is a curious coincidence that in Table II the mean of the average differences in columns 1 and 2 only is the same as that in columns 1, 2, 3, 6, 7, 8, viz. 2·495.

But even allowing for the deformity caused by the droop of the ill-developed and distorted supra-orbital ridge (figs. 6, 7, 8, 9), it appears to me that the whole cavity is diminished in capacity (figs. 10 and 11).

Turning to the question of the share borne by the *soft structures* of the orbital margin in this diminution (see Table I); the measurements of the heads with soft parts were more difficult, and are thus less accurate than the others, but it may be assumed that the figures for the two sides are relatively not far from the truth. I need only refer to the averages. The average difference in the antero-posterior direction is 4, in the vertical 3·35. The mean of these two is 3·67. Regarding only heads 3, 4, and 5, so as to have a strict comparison between wet and dry, the figure is 3·540 *wet*, and 2·495 *dry*. Difference 1·045. This figure 1·045 represents the average deficiency in the anophthalmic orbit due to the soft parts (see Table III).

It thus turns out that the greater share of the faulty development (2·495 mm.) rests with the bone, and the lesser (1·045 mm.) with the soft parts, which is contrary to expectation.

Taking it as definitely proved that in the rabbit removal of an eye in youth results in deficient and irregular growth of certain bones of the skull and face, what is to be said regarding the probability of similar changes in the human subject?

In the rabbit the supra-orbital ridge is a more or less isolated thin plate of bone overhanging the orbit, not likely to be influenced by parts other than the eye, although indeed, the post-orbital process gives attachment to the strong attrahens aurem muscle. In the human subject it forms a more solid portion of the frontal bone, which is itself in relation with the cranial contents, and likely to be influenced as much by the growth of the cerebrum as by that of the eye.

We might expect, therefore, that in the rabbit, as compared with man, the diminution of the vertical diameter would be excessive.

Table II (columns 1 and 2) on the whole confirms this expectation, for 2·66 is a greater proportion of the average total height on the normal side (20·63) than is 2·33 of the average total length on the normal side (25·10).

I cannot think of any such difference between rabbit and man to account for the loss in the antero-posterior direction, and I should suppose that since the antero-posterior orbital length is diminished in the rabbit, the corresponding measurement is likely to be diminished in man, a remark that applies also to the orbital depth.

Lastly, as to the comparative age of operation. The rabbit's average life may be taken as seven years, or 2555 days. Taking sixty years or 21,900 days as the average life of human beings who reach maturity, we find that, by simple proportion, 171 days or about six months corresponds to twenty days in the rabbit. That is, an operation at the twentieth day corresponds to an operation on a child of six months.

But calculation by simple proportion does not take account of the fact that the rabbit matures relatively sooner than the human being. The former is sexually mature at six months, or one fourteenth of its life; the latter at, say, twenty years, or one third of his life.

Looking at it in this light, a rabbit operated on at twenty days corresponds to a child operated on at between two and two and a half years.

The conclusion, then, is that in certain animals the growth of the orbit is influenced by the presence of the eye. Whether the same holds good in man and animals, such as dogs and cats, and the apes, is at present a matter of inference. Personally, I can hardly conceive that such a markedly positive result in rabbits is compatible with a negative result in the others.

TABLE I.—*Heads* 1, 2, 3, 4, *and* 5 (*Wet*, i.e. *with Soft Parts, except Skin*).

	1. Antero-posterior measurement, from anterior angle of orbit to tubercle posteriorly. Inside measure.			2. Greatest vertical measurement. Inside measure.			3. From top of orbit to posterior cusp of last molar tooth.			4. From mid-line of head between the orbit to posterior cusp of last molar tooth.		
	N.	A.	D.	N.	A.	D.	N.	A.	D.	N.	A.	D.
Head 1 . .	21·5	16·5	5	20·5	16·5	4	32·5	29·5	3	31	31	0
Head 2 . .	20·5	17	3·5	19·5	16·5	3	32	29·5	2·5	30	30	0
Head 3 . .	20	16	4	19·5	16	3·5	Lower jaw present.			Lower jaw present.		
Head 4 . .	19·5	16	3·5	19·5	16·5	3	33	30·5	2·5	31·5	31	0·5
Head 5 . .	20·5	16·5	4	20·25	17	3·25	32·5	30·5	2	31	30·5	0·5
Average . .	20·4	16·4	4	19·85	16·5	3·35	32·5	30	2·5	30·87	30·62	0·25

Note.—N. = Normal orbit. A. = Anophthalmic orbit. D. = Difference between N. and A. All measurements are in millimetres.

TABLE II.—*Heads* 3, 4 *and* 5 (*Dry, viz. Soft Parts removed by Maceration*).

	1. Antero-posterior measurement, from anterior angle of orbit to tubercle, posteriorly. Inside measure.			2. Greatest vertical measurement. Inside measure.			3. From top of orbit to posterior cusp of last molar tooth.			4. From mid-line of head between the orbits to posterior cusp of last molar tooth.		
	N.	A.	D.	N.	A.	D.	N.	A.	D.	N.	A.	D
Head 3 . .	25·2	22·2	3	20·6	17·7	2·9	30·5	26·8	3·7	29·6	28·6	1
Head 4 . .	24	22·8	1·2	20·5	18·3	2·2	31·6	29·3	2·3	30	29·4	0·6
Head 5 . .	26·1	23·3	2·8	20·8	17·9	2·9	32	29·7	2·3	30·6	30·1	0·5
Average . .	25·10	22·76	2·33	20·63	17·96	2·66	31·36	28·6	2·76	30·06	29·36	0·70

	5. From mid-line of head between orbits, at right angles to it, to the superior orbital margin.			6. From anterior junction of zygoma with temporal process of malar bone, to bottom of post-orbital fissure.			7. From the same point to the bottom of the supra-orbital notch.			8. Approximate orbital depth.		
	N.	A.	D.	N.	A.	D.	N.	A.	D.	N.	A.	D.
Head 3 . .	11·3	11·3	0	18 0	15·4	2·6	20·5	18	2·5	17·5	15	2·5
Head 4 . .	10·1	11·3	+1 2	19·2	17	2·2	21·8	19·8	2·0	18·1	15·8	2·3
Head 5 . .	11·1	11·1	0	20·7	17·8	2·9	20·9	18·3	2·6	18·5	16·4	2·1
Average .	10·83	11·23	+ ·4	19·30	16·73	2·56	21·06	18·7	2·36	18·03	15·73	2·30

Mean of average differences in Columns 1, 2, 3 and 6, 7, 8 = 2·495.
„ „ „ „ 1 and 2 only = 2·495.

Note.—N. = Normal orbit. A. = Anophthalmic orbit. D. = Difference between N. and A. All the measurements are in millimetres.

TABLE III.—*Comparison of Heads* 3, 4, *and* 5 *in the Wet and Dry States to show the Relative Failure of Development of Bone and Soft Parts* (*Vertical and Antero-posterior Measurements only*).

Mean of the average antero-posterior and vertical differences, in millimetres	WET =	3·540, which is the average difference in length and height due to BONE + SOFT PARTS.
Ditto, ditto . .	DRY =	2·495, which is the average difference in length and height due to BONE only.
Subtract DRY from WET.	=	1·045, which is the average difference in length and height due to SOFT PARTS only.

Postscript.—I have refrained from speculation as to the cause of the failure of growth, and have left this question entirely open. It is interesting to remember, however, that the post-orbital process is malformed *and drawn towards the orbital cavity* on the anophthalmic side (Figs. 6, 7, and 11), in spite of the strong pull of the attrahens aurem muscle in the opposite direction.

Dr. Thomas H. Bryce has pointed out, after an anatomical examination of the skulls, that the uniformity of the developmental failure in all the bones entering into the formation of the orbit is very marked. (*November 8th*, 1900.)

The PRESIDENT said one practical result of the paper to ophthalmic surgeons would, perhaps, be that when it was necessary to remove an eye from a very young child they would be more than ever anxious to introduce an artificial eye as soon as possible. Parents frequently objected to that on account of the possible danger of fracture of the artificial eye, especially if it were made of glass. Perhaps in those cases celluloid would be preferable to glass, or some india-rubber material possibly better still. He would be glad to hear the opinions of any members.

Dr. GRUBER thought that importance should be attached to the fact that there was atrophy of the ocular muscles, because their insertions to the eyeball were detached.

That fact would necessarily have some influence on the size and shape of the orbit.

Dr. Thomson, in reply to Dr. Gruber, said his remark might be true, but his paper was devoted to ascertaining the facts, not entering into the question of causation.

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4. *On so-called paresis of divergence.*

By G. A. Berry, M.B.

Although I have seen a few similar cases, I shall, in order to be as brief as possible, base my remarks on the following case:

J. A—, æt. 44 years, a previously healthy man, who neither smokes nor drinks, suddenly developed a diplopia on the morning of the 16th September last. He is employed on night duty at the Edinburgh gasworks, principally in regulating pipes, meters, etc. His family history is good and unimportant.

He was first seen by me at the Royal Infirmary three days after the onset of the diplopia. He stated that in the interval he constantly saw double. In the mornings, according to his wife's statement, he had quite a marked convergent squint. This passed off in the course of the day after rest. Being always at work at night, he slept during part of the day.

On examination, there was no apparent interference with the associated lateral or vertical movements of the eyes. On distant fixation there was manifest convergence.

In addition to this history of a marked manifest squint, which existed in the morning and afterwards diminished to such an extent as to be unnoticeable, the further examination brought out several points of interest.

The convergence on distant fixation, tested with a candle flame and red glass in front of one eye, equalled

about 3 metre angles (*i. e.* convergence to a point lying about $\frac{1}{3}$ metre or 13 inches from the eyes). Any abnormal convergence was not particularly apparent on mere inspection. The diplopia was corrected by a prism of 22°. At the reading distance there was much less excess of manifest convergence; indeed, there did not seem to be constantly any diplopia at all. Diplopia could always be elicited, however, with the flame and red glass. Yet there existed a high degree of latent convergence. Either eye, if occluded, at once deviated inwards in a most marked manner. Tested for objects at the distance of six to eight feet, the double images were found to be considerably closer together on fixation to either side than on looking straight forwards. The latter separation was also somewhat less when the eyes were turned upwards, and somewhat greater when the eyes were turned downwards. The power of fusion was good. The diplopia could be corrected by the same prism at different distances, provided the object fixed was not too rapidly moved, either towards or away from the patient. This was even found to be the case for the fixation of a candle flame with a red glass in front of one eye.

The treatment consisted in complete rest, with bromide of sodium. After four days all spontaneous diplopia had disappeared. There was still some latent convergence, though very much less than formerly. Diplopia could also be elicited with a red glass, and this was corrected by a prism of 12°. After a week only a trace of latent convergence could be made out by means of a vertically refracting prism. The patient then returned to work, and there has since been no recurrence of the symptoms.

The case was evidently not one of the sudden manifestation, owing to abolished or restricted fusion power, of a previously existing latent convergence. This cause, although in my experience far from frequent, undoubtedly accounts for the sudden appearance of some concomitant squints. I have seen this, however, only in young people. The good fusion existing throughout, and the eventual

loss of latent convergence, clearly pointed to some other cause. The character of the diplopia again showed that no paresis of any of the ocular muscles existed. The diagnosis was therefore restricted to either *convergence spasm* or *divergence paresis*. This was, in fact, an example of an affection which has recently received some attention, and been generally described as *paresis of divergence*.

In some respects it might seem immaterial whether such cases are regarded as spasm of convergence or as paresis of divergence. The effect on convergence might well be considered to be the same in either case. Yet it is not only of interest, but surely of some clinical importance, to attempt to make a distinction between a state of irritation leading to the stimulation of some nerve-centre, so that its regulation is withdrawn from normal control, and a curtailment of power by which, notwithstanding the existence of a normal impulse, the effect falls short of what takes place under conditions of health.

A curious feature in these cases is the diminution in the distance apart of the double images on lateral fixation. Evidently if both external recti were paretic, the images, instead of being nearer together on looking to either side, would be further apart. The fact that the reverse is the case has been considered as diagnostic of paresis of divergence. Certainly, on the assumption that there is any weakness in the innervation of the external recti, a weakness in their lateral associated contraction must from this symptom be excluded. This fact seems, however, to be the only ground for assuming that a weakness in their divergence innervation must therefore exist. Before offering an explanation for this relation of the double images on lateral fixation, it is necessary first to consider a point which is mainly mechanical. We must inquire what must be the double image relations on the assumption that the same degree of over-convergence is maintained as the eyes move from side to side. Does the same approximation of the images on lateral fixation, as characterises the affection with which we are now dealing, result as a consequence merely

of concomitance? It is easy to show that this is not so. Let O (Figs. 24 and 25) be an object in the middle line. Let us suppose, that in attempting to fix O, it is impossible to do so binocularly owing to the tendency to over-convergence. One eye alone is then used for fixation, while the other deviates, in a degree corresponding to the over-convergence. Suppose the left to be the fixing eye; the visual axis of the other then cuts the line of fixation at c. If the object be moved to O' both eyes must move laterally through the same angle ϕ, on the assumption that the same degree of convergence is maintained between their axes. The left being still the fixing eye, their axes would now meet at c'.

In the triangles Rmc and Lmc' the angles ϕ and m are equal, therefore the angles c and c' are also equal. In the triangles LRc and LRc', the angles c and c' being equal, it follows that L, R, c, and c' are points on a circle—the circle of equal convergence. Now prolong Rc and Rc' till they meet the line or circle along which the fixation object is moved at ω and ω'. Then join $O\omega$ and $O'\omega$. We have then two angles θ and θ' which are related to each other in the same manner as the angular excentricities of the misdirected right eye's retinal images, for fixation in the middle line and laterally, respectively. If, therefore, the angles θ and θ' are unequal, the images of O and O' will lie at different distances from the fovea of the right eye and be projected accordingly upon the fixation plane or circle, the one further than the other from the true image of the left eye.

The figures enable us to demonstrate geometrically the relation $\theta : \theta'$ in a general way.

The angle a' is less than a, because O' lies outside the circle of which LR is a chord and which passes through O. In the triangles RcO and $Rc'O'$ the external angles c and c' are equal to the two internal and opposite angles respectively, $a + \theta$ and $a' + \theta'$. We have then—

$$c - a = \theta$$

and $$c' - a' = \theta'$$

FIG. 24.

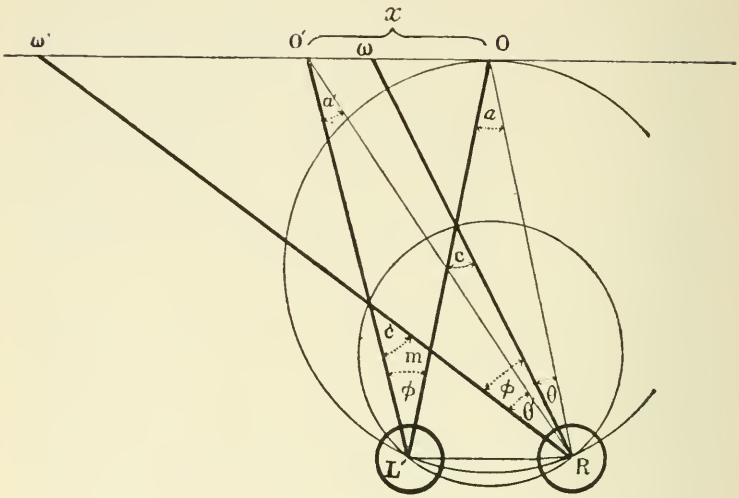
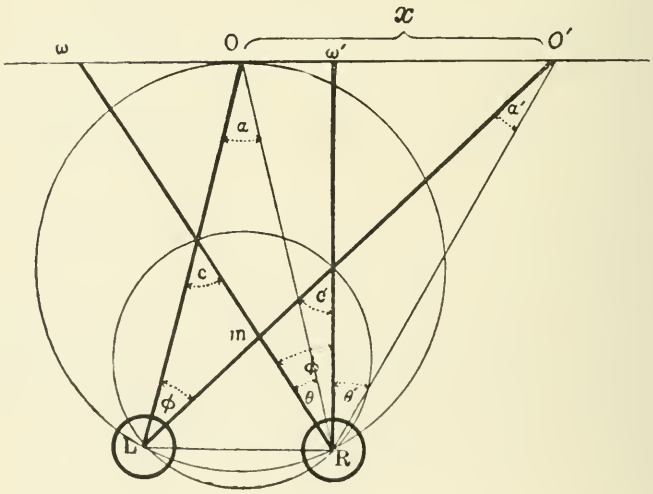


FIG. 25.



but as $c = c'$ and a is greater than a', it follows that—

$c' - a'$ is greater than $c - a$,

i. e. θ' is greater than θ.

The false image in the right eye must therefore be more excentric on fixation to the side than when the object looked at lies in the middle line in front of the eyes. The excentricity, too, will be greater for objects moved along a plane at right angles to the face than for those moved in a circle, as on the perimeter. Only for objects moved along a circle passing through O and the centres of rotation of the two eyes is the excentricity the same in both cases.

A constant degree of convergence should therefore sometimes be associated with a greater and not a less degree of separation of the double images to the side.

It may easily be shown that this difference between θ' and θ is greater the smaller the angle of convergence at c is (*i. e.* the greater the circle of equal convergence) and the nearer the plane along which the fixation object is moved is situated to the circle of equal convergence.

Indeed, only under such circumstances as considerably increase the difference could the greater separation of the double images on lateral fixation be appreciable. For a test made at the distance of ten or twelve feet and more and any marked degree of over-convergence the separation would be practically the same for lateral as for central fixation.

A geometrical proof of this kind, although it enables one to demonstrate the greater excentricity of the deviating eye's image on lateral fixation, does not afford data for the calculation of the increase of excentricity under different conditions, and therefore of how far the extra excentricity is likely to be appreciable. I therefore append a more complete analytical proof for the case, where the object of fixation is moved along a horizontal line in a plane parallel to the vertical plane through the centres of rotation of the two eyes. With the same lettering as in the figures, and putting D for the perpendicular distance from the eyes of the plane along which the object of fixation is moved; d

for half the inter-ocular distance, and x for the variable distance OO', we get—

$$a = 2\tan^{-1}\frac{d}{D}$$

$$\text{and } a' = \tan^{-1}\frac{x+d}{D} - \tan^{-1}\frac{x-d}{D}$$

$$= \tan^{-1}\frac{2dD}{D^2 - d^2 + x^2}$$

$$\text{but } \theta = c - a$$

$$= c - 2\tan^{-1}\frac{d}{D} \quad . \quad . \quad . \quad . \quad . \quad 1.)$$

$$\text{and } \theta' = c - a'$$

$$= c - \tan^{-1}\frac{2dD}{D^2 - d^2 + x^2} \quad . \quad . \quad 2.)$$

From 2.) it is evident that when x increases (*i. e.* when the fixation object is carried further and further away from the middle line) θ' also increases. For $x=0$ (coincidence of O and O') $\theta' = \theta$, as $\tan^{-1}\frac{2dD}{D^2 - d^2} = 2\tan^{-1}\frac{d}{D}$.

1.) and 2.) also show that the proportion $\frac{\theta'}{\theta}$ is greater the smaller c is. The less marked, therefore, the over-convergence for fixation in the middle line, the more appreciable should be the increased separation of double images on lateral fixation.

Whilst there can be no doubt, therefore, that maintenance of a constant degree of convergence would cause a greater separation of the double images on lateral fixation, we must next determine whether the amount of this difference is sufficiently great to be appreciable under circumstances which ordinarily present themselves, and under conditions in which the test is usually made. To get at this we have to calculate the distances along the horizontal line cut by lines from the eye, forming angles θ and θ' with RO and RO', but on the opposite side from those shown on the figures, as these new lines represent the directions of projection.

I have already referred to the conditions which deter-

mine whether the difference on medial or lateral fixation is likely to be appreciable or not. I add here one example. For fixation on a plane about twenty inches distant and convergence towards thirteen or fourteen inches, the double images in the middle line would be rather over an inch apart, and for fixation 30° to the side about $1\frac{3}{4}$ inches. This difference would no doubt be quite appreciable.

Under all circumstances, however, retention of a permanent degree of convergence would at least not cause the images on lateral fixation to appear nearer. If there were any appreciable difference they would, on the contrary, appear further apart.

When, therefore, the images are closer on lateral fixation, it shows either that the right eye image is projected on to a plane nearer to the eyes when a lateral object is fixed than when fixing a central object, or that *convergence has decreased*. The former assumption is one which there does not seem to be any reason for entertaining. We must, therefore, infer that in spasmodic convergence, or so-called paretic divergence, the tendency to over-convergence is not so firmly established, it is incapable of being lessened under conditions in which equally strong convergent movements are not habitually called for.

It is interesting to observe that although, on the assumption of a constant degree of convergence, the excentricity of the image in the deviating eye, of an object fixed by the other eye, may increase, but never diminish, on lateral fixation, this is not the case, if convergence, while always excessive so far as the requirements of binocular fixation are concerned, is altered on lateral fixation, in a manner which may be looked upon as habitual. Let us assume, *e. g.*, that as the eyes move to either side, the degree of convergence diminishes to the extent necessary to maintain the fixation of objects in a plane near to and parallel with the eyes (*e. g.* the page of a book). The relations of the excentricity of the deviating eye's image, when the other fixes an object centrally or laterally placed on a plane parallel to this one, but at a greater distance from the eyes,

may then be deduced from the accompanying figures (Figs. 26, 27). Here, although there is some difference according to which of the two eyes fixes to either side, we have $\theta = A - a$ and $\theta' = A' - a'$. For anything beyond ten feet distance of fixation, at any rate, a and a' are so small that they may be neglected. Practically therefore θ then $= A$ and $\theta' = A'$. And as A is always, on looking to the side of the fixing eye, and also, except for small lateral displacements on looking to the other side, greater than A', θ is generally greater than θ'. With any ordinary degree of over-convergence the diminution of θ' for distant fixation (ten feet or more) would be readily appreciable. On fixation 30° to either side, the double images would appear distinctly closer together than on median fixation.

With the same notation as before, and putting D' for the vertical distance of the nearer plane from the line joining the rotation centres of the two eyes, we get as the general value of θ' for varying values of x:

$$\theta' = \tan^{-1} \frac{2dD'}{D'^2 - d^2 + \left\{\frac{D'}{D}\left(x \pm d\right) \mp d\right\}^2} - \tan^{-1} \frac{2dD}{D^2 - d^2 + x^2} \cdot \quad 3.)$$

On differentiating the two angles in 3.) with respect to x it is found that the rate of change with increase of x is much greater in the first (A') than in the second (a'). Approximately,

$$\frac{\frac{d}{dx}A'}{\frac{d}{dx}a'} = \frac{D}{D'}$$

The maximum value for θ' is therefore got for the value of x which makes $\frac{D'}{D}(x \pm d) \mp d = 0$, *i. e.* $x = \pm d\left(\frac{D}{D'} - 1\right)$. This is also evident from the figures, as then $A' = \tan^{-1} \frac{2dD'}{D'^2 - d2} = 2 \tan^{-1} \frac{d}{D'}$.

In fixing with the left eye then, for instance, if the fixation object be carried to the left, θ' diminishes constantly

FIG. 26.

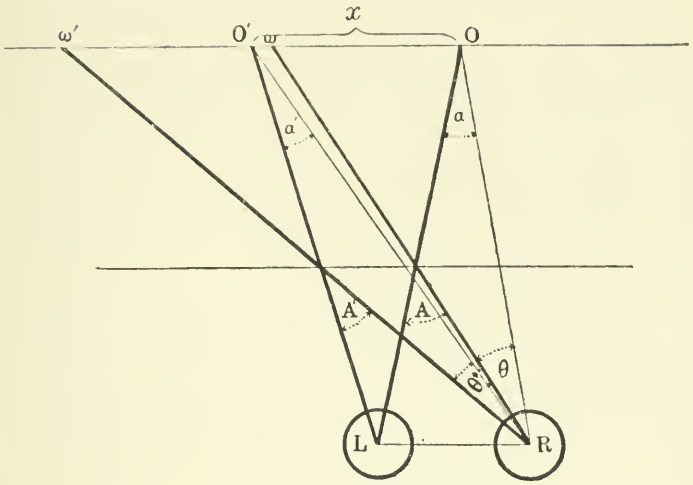
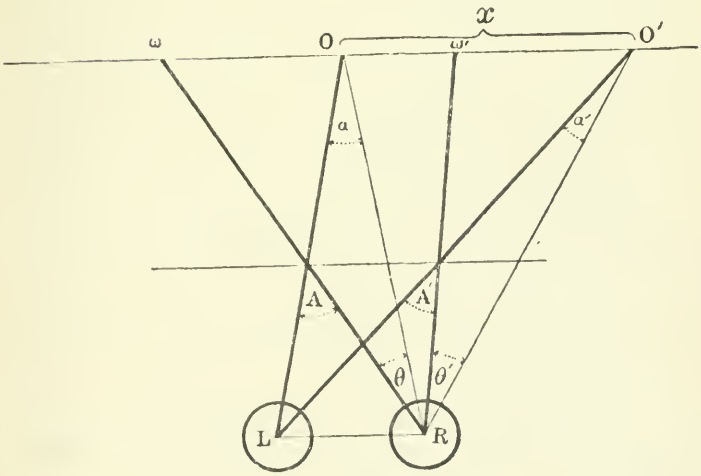


FIG. 27.



as x increases, while if the object of fixation be carried to the right θ' first increases until $x=d\left(\frac{D}{D'}-1\right)$, and then diminishes, and *vice versâ* for fixation with the right eye.

An equal change in the angle to either side, and a consequent equal change in the degree of separation of the resulting double images, would only be got when one eye fixes to one side and the other to the other side, while the greatest and continuous decrease of separation would take place when the right eye fixed objects to the right, and the left eye objects to the left, probably a less frequent condition than the opposite, which is the rule in ordinary alternating concomitant strabismus.

It seems not improbable, when we take into consideration the fact of the approximation of the double images on lateral fixation in cases like the one here recorded, that such a retention of the habitual association which occurs between convergence and lateral fixation is actually, or at all events approximately, what takes place.

This consideration surely, too, points to *spasm of convergence* as the cause. We can readily understand that the effect of an irritation of the converging centre might be to withdraw the relaxation of convergence beyond a certain point (a point which may, and no doubt does, vary from time to time) from the control of the will, and yet to leave the play of convergence, as the eyes move from side to side, to be effected in a habitual manner. This would be similar, in fact, to the natural convergent movements which would take place if, on attempting to fix a more distant object moved from side to side, the eyes kept converged on points in a plane which lay nearer to them. This, while consistent with the idea of spasm, appears to me a much less likely state of matters to co-exist with anything of the nature of paralysis.

I have already stated that there was no obvious reason why the peculiar relations of the double images should to some appear in favour of the diagnosis, paresis of divergence. If they have any bearing at all on the diagnosis,

it seems more reasonable to infer that they suggest spasm of convergence.

In support of such a diagnosis, however, it is perhaps better to rely upon arguments afforded by the clinical facts which are afterwards referred to.

The effect of habit, indeed, often asserts itself more or less even where the conditions are peculiarly unfavourable. Just as we often see in the case of abducens paresis that the resulting double images are further apart on downward than on upward fixation, because of the habitual association of convergence with downward rotation and the absence of any call for convergence when the eyes are directed upwards, so we find a degree of abnormal convergence lessen when associated with wide lateral movements, as there is not habitually as great a degree of convergence associated with marked lateral fixation as for central fixation. In the case here referred to, both these effects of habit were evidenced.

It has sometimes been questioned whether the increase of convergence on looking downwards, which is common, more or less, to all forms of pathological convergence, may not be dependent solely upon anatomical conditions. That this is *not* the case I have shown by an examination of a number of individuals as to the influence of downward and upward rotation of the eyes on the degree of what may be looked upon as physiological latent lateral deviations. The result of this investigation was communicated to this Society. The cause is therefore, no doubt, as was first maintained by v. Graefe, the influence of habit.

The question as to whether this curious and rare form of strabismus is the result of convergence spasm or of divergence paresis is not, perhaps, one which the consideration of such cases alone can enable one to decide. As the symptoms might be supposed to be the same in the one case as in the other, the proper conclusion to draw, apart from other considerations, is, as it appears to me, that the diagnosis divergence paresis is not justified. Such a diagnosis involves the admission of a divergence

innervation which for many reasons is, at least, problematical. I have seen the same condition of suddenly arising convergent strabismus followed, after a short time, by a persistent associated lateral deviation of the eyes of an undoubtedly spasmodic nature, which has also disappeared after some time. One case of this kind I communicated to this Society. Such an association is more than suggestive of spasm.

In the present case the very curious great excess of latent over manifest convergence for near fixation would also seem to point to spasm as the cause of the symptoms. On accommodation for a near object, with one eye occluded, the tendency evidently existed to association with an undue degree of convergence. This, too, was presumably the result of an abnormally excitable state of the convergence centre. It was, however, counteracted by fusion the moment binocular fixation became possible.

This tendency to overaction is also seen sometimes in what is known as spasmodic myopia. In my experience there is no such thing as a constant spasmodic myopia. The nature of such cases is this: that whenever attempts at accommodation are made they result in a degree which is in excess of the requirements. Often the subjects are slightly hypermetropic, and therefore have to accommodate to see smaller objects at a distance. This tendency is probably a purely innervational one, and seems analogous to the excessive latent convergence in the case which I have just described.

Apart from the above considerations, which point to spasm of convergence instead of paresis of divergence, there are, as I have elsewhere pointed out, many reasons for altogether disbelieving in a real divergent innervation, *i. e.* one starting from what, from analogy, we might call a divergence centre.

The common conception seems to be that if one admits the existence of a convergence centre there must surely be a divergence centre as well. There is a centre for associated eye movements to the right, and one for movement

to the left, and these are antagonistic. And, indeed, all analogy would point to the arrangement of innervation centres in antagonistic pairs. I long ago pointed out that there were physiological and clinical reasons for rejecting the view that the cessation of convergence was brought about by *active* divergence, and therefore for concluding that no diverging centre existed.

Divergence would thus constitute an exception to the rule which obtains in the body generally, as well as an apparent exception to what takes place in regard to the lateral movements of the eyes. I say apparent, because the relative divergence which takes place on the cessation or relaxing of a converging impulse is no doubt effected in the same way as is the return of the eyes to the middle line on the cessation of an impulse which causes them to rotate to one side. The divergence on the one hand is due to the passive return of the external recti to the length, which corresponds to their tonic state of innervation after the degree of greater stretching to which they have been subjected is removed. The return to the middle line of eyes laterally displaced, on the other hand, is due to the passive return of the associated internus of one eye and externus of the other, to physiological states which they have in virtue of their tonic innervation. An active innervation is not called for until rotation beyond the middle line in the opposite direction is begun.

This view of the matter was also entertained by the late Professor Alfred Graefe. In his admirable monograph on the anomalies of the ocular muscles in the second edition of the *Graefe-Saemisch Handbook*, he says:—"One can only agree with Berry when he rejects as superfluous the assumption of the existence of a special divergence innervation, and consequently of a definite centre for this innervation."

I shall content myself by repeating here only two arguments which can be adduced against the existence of a divergence centre. It is evident that divergence beyond parallelism of the axes is never a physiological

requirement, and therefore that the only imaginable use for a divergence innervation could be to cause the return of the eyes to a position of parallelism from that of convergence. Like convergence, divergence must be primarily regulated by fusion, and as fusion is not called for with divergent axes, no habit of absolute divergence can be naturally acquired. Yet it surely ought to be possible to induce further divergence in the interest of single vision by means of abducting prisms were there an active centre for divergence. In the case of convergence, one finds that very little practice is required to overcome adducting prisms of a strength which cause a very much greater degree of convergence than is ever called for physiologically. No doubt abducting prisms can also be overcome, so that it *is* possible to have an absolute divergence. But there is a limit to the degree of this absolute divergence even after prolonged exercise with prisms. This limit coincides with the degree of divergence, which is assumed when there is a complete loss of any converging power. In other words, the limit of possible divergence is the same as the starting-point of convergence. This is the physiological reason for disbelieving in a divergence centre.

A more important, more convincing, evidence is afforded by certain conditions of paralysis. For instance, cases occur in which there is a complete loss of power of associated movement to one side, or even (though these are extremely rare) to either side, without any interference with convergent movements and with return to parallelism on convergence being relaxed.

It might be argued, however, that just as the interni, though not receiving an innervation to lateral associated movements, are able to respond to a convergence impulse, so the external recti might retain the power of active contraction in response to an unimpaired divergence impulse.

This loophole of escape for those who cling to a divergence centre is surely closed, however, by the not

unfrequent co-existence of complete paralysis of one externus with the power of bringing the affected eye back to the middle line on the cessation of convergence. In such cases the same result follows the relaxation of an active contraction of the internus in association with the externus of the other eye. In neither case would the mere removal of the internus innervation effect the return of the eye to the middle line, while the absence of any power in the externus makes it certain that it responds to no active innervation which is antagonistic to that which is supplied to the internus. The return of the eye to the middle line must therefore have a purely physical cause, or must depend upon *the resumption by the muscle of that state or that length which corresponds to its tonic innervation when opposed by the tonically innervated internus.* That the latter, and not the former, is the true cause, seems to me evident from the phenomenon of so-called secondary contracture of the antagonist in cases of ocular paralysis. This phenomenon may either slowly develop or be apparent from the outset. It is no doubt due to *the loss, gradual or sudden, of this tonic innervation in the paralysed or paretic muscle.* It is not due to any change which takes place in the innervation or in the physical state of the antagonist. With the more or less complete disappearance of tonic innervation in an external rectus, for instance, a greater or less preponderance of action takes place in the internus whose tonic innervation is unimpaired. Thus is developed the position of convergence, which in many cases characterises the position of rest in paralysis or paresis of the sixth nerve. The term secondary contracture is therefore, I believe, and have always maintained, a misnomer. It is one of the many terms expressive of conceptions borrowed from muscular changes in other parts of the body. It is very generally, I think, lost sight of that the eye muscles differ in various ways, both anatomically and physiologically, from the skeletal muscles.

In conclusion, I may say that I have brought forward this case—a typical example of what in my experience is a

rather rare affection—first, to prove that the nature of the diplopia is an evidence of the pathological convergence being diminished on lateral fixation. Again, I wished to offer an explanation of this symptom. As I have explained, it can only be caused by the influence of habit. Finally, and more particularly, I wished to make it clear that *the correct diagnosis is spasm of convergence* and not paresis of divergence, any more than a persistent associated lateral deviation of both eyes is to be looked upon as an expression of paresis of associated movement in the opposite direction. Both conditions, moreover, are, in my experience, always transitory, whereas both paresis of convergence and of associated lateral movement are often permanent.

(*January* 31*st*, 1901.)

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5. *On two rare forms of non-paretic non-concomitant squint.*

By G. A. Berry, M.B.

(1) *Strabismus fixus.*—I have met with four cases of the peculiar and, apparently, congenital condition to which the above name was first given, I believe, by v. Graefe. One eye is forcibly directed inwards, and apparently more or less firmly fixed in this position. Tenotomy of the internal rectus fails to effect any improvement. Even a very free separation of the indirect attachments of the muscle produces little effect. The eye is evidently bound down in its abnormal position by extensive and deep-seated connective-tissue attachments. The exact nature of these attachments is not known, so far as I am aware.

In all my cases I have made the diagnosis only after operation. Each case was diagnosed at first as a congenital complete externus paralysis, with preponderance of the internus. And yet the differential diagnosis ought to be easy enough. It is to this point of diagnosis that I wish

to direct attention. In any case where a doubt might exist as to whether the condition were a purely paralytic one or one of fixed strabismus, all that is required to establish the diagnosis is to test whether the eye can be rotated outwards with fixation forceps. No difficulty is experienced in doing this in the case of paralysis. In strabismus fixus, on the other hand, the eye may by traction be displaced outwards, but any attempt at rotating it fails, and only presses it back into the orbit. Any one who has had occasion to operate in these cases will have experienced the impossibility of bringing about a real rotation.

(2) *Strabismus due to defective associated lateral movements of the eyes.*—I have recently met with a case which, in my experience, is unique. At all events, although I have seen slight degrees of the same defect as a latent deviation, I have never met with such a marked example in a manifest form.

The patient was a little boy, æt. 8 years, sent to me from the north of Scotland on account of squint, with a view to operation. On examination, I found that when the boy looked forwards there was complete parallelism of the axes of his eyes. He also possessed the power of converging naturally, and had true binocular vision. The eyes were practically emmetropic, and the visual acuity normal in each.

On looking to the right the left eye turned upwards and inwards to such an extent that the cornea was almost completely covered by the upper lid. Exactly the same thing happened on looking to the left. Here the right eye, instead of following the left, turned upwards and inwards in the same excessive degree, so as to give rise to an unsightly convergent and sursum-vergent squint.

This abnormal association apparently began the moment the lateral movements were called for. At all events the squint was most evident for fixation at 10° to either side. The child was consequently as often squinting atrociously as not. Yet there was no diplopia.

Obviously the case was not one for operation. I ordered

a pair of spectacles with blinker sides, which necessitated rotation of the head on looking sideways. With these he comparatively seldom squinted during the short time he was under observation afterwards. I cannot say, however, whether they have had the effect of checking the faulty association.

What is the prognosis in such cases? I have referred to it here, as I am in any case discussing some rarer forms of squint in the hope that members of this Society may have had opportunities of observing similar cases for a longer time.

The slighter degrees of the same defect appearing in a latent form are not so uncommon. It is therefore unlikely that cases such as I have described are altogether unknown. (*January* 31*st*, 1901.)

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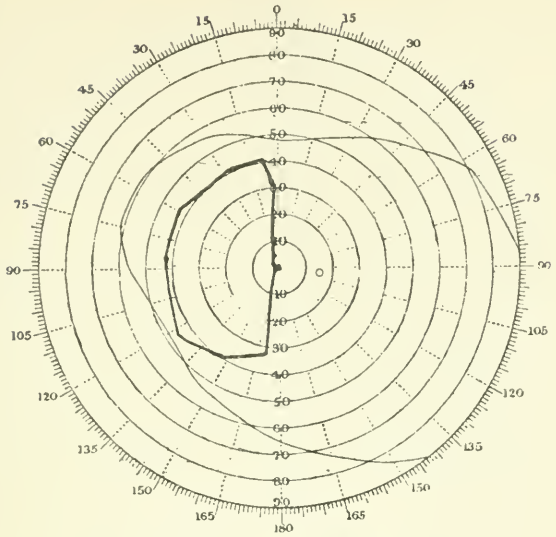
6. *Double temporal hemianopia with slight optic neuritis and hemianopic pupil reaction. ? Hæmorrhage into optic nerve-sheath at chiasma. ? Gumma.*

By A. Hugh Thompson, M.D.

A. B—, married woman, æt. 24 years, came to the Western Ophthalmic Hospital on April 24th, 1901, and stated that sixteen days previously, after a violent attack of retching at five o'clock in the morning, while in bed, she had partially lost her sight, it having been fairly good previously.

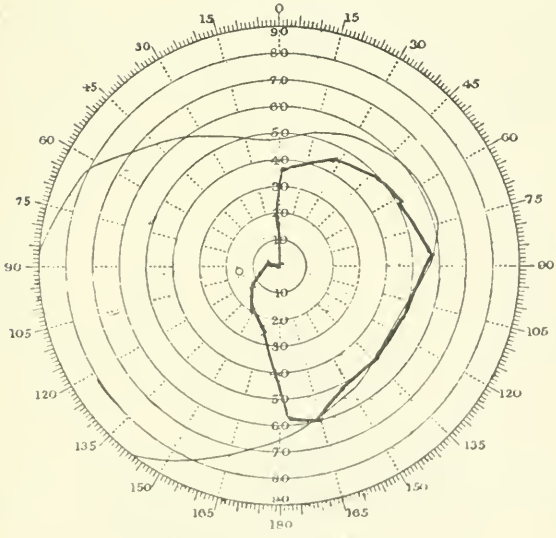
On examination, both eyes appeared to be totally blind in the outer half of each field, retaining some vision, but less than $\frac{6}{60}$, in the inner half. Both pupils were dilated, and neither contracted on light being thrown in from the outer side, but contracted readily on light being thrown in from the inner side. Each cornea was to some extent

FIG. 28.



Right field of vision, May 5th.

FIG. 29.



Left field of vision, May 5th.

nebulous, the right only slightly, the left more so. On ophthalmoscopic examination, the right disc appeared decidedly hazy, the rest of the fundus normal; the left disc and fundus appeared distorted, owing to the corneal nebula; but the margin of the disc was fairly clear, and there was probably no neuritis. There were no hæmorrhages in either fundus. Owing to the immobility of the pupils to light on the outer side, it was never necessary to employ a mydriatic for ophthalmoscopic examination.

"The patient had one child six years ago, who died at the age of six months. Since then she had not been pregnant up till now, when she is told by her doctor that she is pregnant, although her monthly periods have not ceased. She has had a good deal of retching lately, but no headache. She has noticed numbness and pain in her fingers lately sometimes, especially the second finger on each hand. She has also suffered a great deal from thirst of late, and passes a great quantity of water." (Urine, subsequently tested, was found to be free from sugar and albumen; specific gravity 1003.)

The fields of vision in each eye taken by McHardy's perimeter were found at the first visit to be bounded by a vertical line passing through the fixation point. The refraction, estimated a fortnight later under homatropine and cocaine, was:

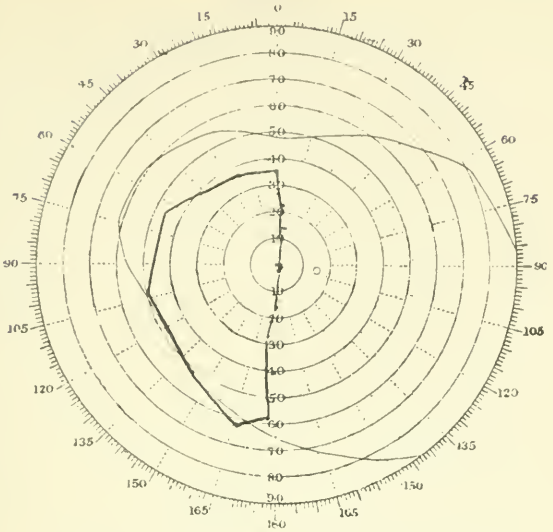
Vision: R. $-$ 2·0 cyl., axis \ = fingers.

L. $\frac{-\ 0{\cdot}5}{-\ 2{\cdot}0\ \text{cyl.}\ 70^{\circ}} = \frac{6}{60}$.

Treatment:—Mist. Pot. Iod., gr. v, t. d. s., produced nausea and was discontinued. Pil. Hyd. c. Cret., gr. j, t. d. s.

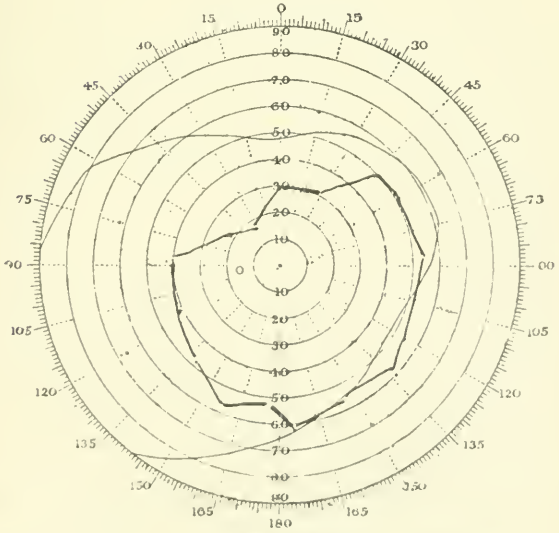
(*May 2nd*, 1901.)

FIG. 30.



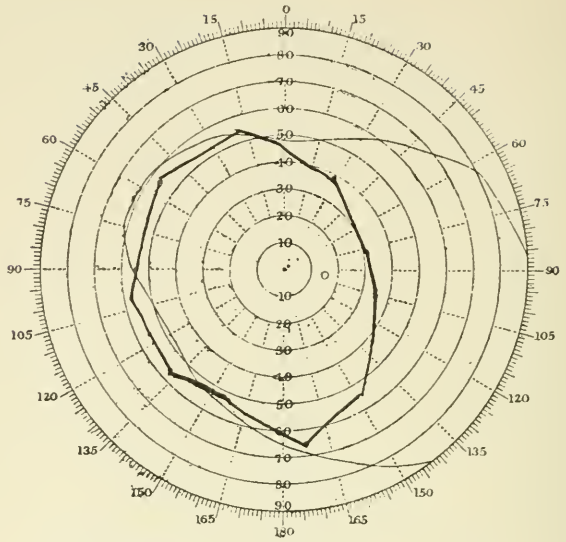
Right field of vision, May 29th (2 mm. square).

FIG. 31.



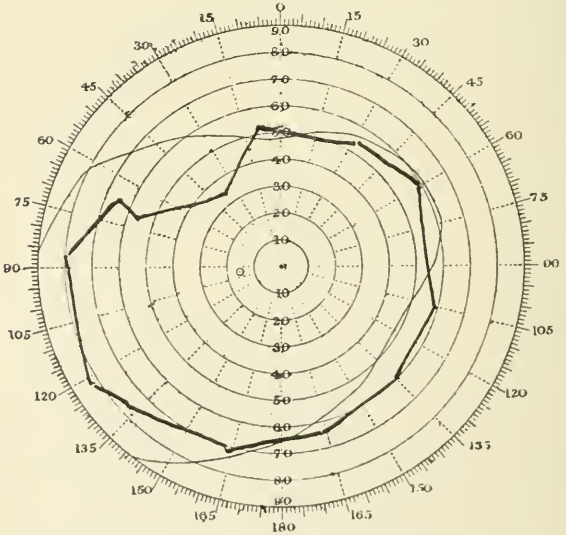
Left field of vision, May 29th (2 mm. square).

FIG. 32.



Right field of vision, June 27th (10 mm. square).

FIG. 33.



Left field of vision, June 27th (10 mm. square).

Subsequent changes.—The *optic neuritis* in the right eye remained well marked for two or three weeks, and then gradually became much less marked. The left fundus did not change. The *hemianopic pupil reaction* very soon showed a marked diminution in the *left* eye, but in the *right* was still well marked on May 29th, and present to some extent on June 11th.

The *field of vision* in the *right* remained strictly limited to the inner half on May 29th (see Fig. 30), but on June 11th was already encroaching on the outer half, and on June 27th (see Fig. 32) was still advancing. In the *left* eye it included the fixation point as early as May 5th, so that it is possible that the vertical line of demarcation found at the first examination was fallacious. (In the right eye the vertical line was found on so many different occasions that there could be no doubt about it.) The left field then steadily increased, so that by June 27th (see Fig. 33) it was nearly full, except for a small sector up and out.

The *vision* of both eyes improved *pari passu* with the fields, the right improving to $\frac{6}{60}$, the left to $\frac{6}{18}$.

General condition.—The pregnancy became unequivocal. Occasional retching, thirst, and polyuria, with complaints of numbness of fingers, continued, varying from time to time. On June 27th, for the first time, was noticed an ulcer at the back of tongue (left side) with enlarged gland below jaw.

P.S. (August 3rd, 1901).—The right eye still shows hemianopic pupil reaction, disc becoming atropic, V. $\frac{6}{24}$. Left eye normal, V. $\frac{6}{12}$. The ulcer on the tongue is unhealed, and the gland still to be felt. There is a further appearance pointing to tertiary syphilis, *viz.*, a swelling over second rib on left side, near its junction with the sternum, semi-solid, but with deep fluctuation. It was first noted on June 15th, but has considerably increased in size. Patient has been taking mercury for three months, and has been given Pot. Iod. and Sod. Iod. at intervals, but as

yet has taken hardly any, as they always make her retch. She is in the fifth or sixth month of pregnancy.

P.P.S. (August 9th, 1901).—Dr. Kellock (Middlesex Hospital) pronounces the lesions on tongue and chest wall to be syphilitic, the former a mucous tubercle, the latter a gumma of periosteum.

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7. *Paralysis of third nerve, with unusual complications.*

By P. H. Mules, M.D.

A man, æt. 75 years, suffering from headache and general malaise, applied carriage varnish and an irritant liniment to the right side of the scalp and forehead. Severe inflammation with deep sloughs of small patches of tissue followed, together with a comatose condition and respiratory interference. With treatment, the cerebral symptoms and inflammatory œdema of the scalp passed away; then it was found that paralysis of the third nerve had come on.

The malaise and headache were thought to be antecedent to herpes zoster of the ophthalmic division of the fifth. The powerful irritants masked the vesicular eruption, so that its existence could only be surmised.

The third nerve paralysis, the prominent symptom, occasionally, though rarely, accompanies zoster of the fifth, so a hopeful prognosis was given; four months later the affected muscles had regained their normal movements.

Notes of case by his ordinary medical attendant.

May 5th, 1900.—Called to see a person suffering from gastritis caused by drinking new champagne. Patient was a healthy man of seventy-five years, who had spent many

years in South African exploration. Many years ago had run a thorn into left eye, and some years afterwards had a gunshot accident to right eye. Previous to seeing him patient had taken about half a box of Page Woodcock's Wind pills, two ounces of salts, a few Carter's Little Liver pills, and then, finding himself no better, two ounces of tartar emetic. As soon as the vomiting had ceased, he was put on bismuth and soda mixture.

6th.—Vomiting ceased; is a good deal better.

7th.—Owing to his headache he applied to his scalp and forehead a preparation called "Coachaline," for "saddles, carriage tops, harness, boots, and all other kinds of leather, rendering it waterproof, soft, and pliable; also a healing ointment for scratches and sores on horses and cattle, and has no superior as a hoof ointment." This he left on all night, and then, finding his head worse, wiped it off.

8th.—Considerable pain in head, whole of right side of scalp in a condition of acute inflammation. Right eyelid œdematous. Temp. 100° F.; pulse 110. Patient had a bad night, rambling in his sleep.

9th.—Noon, sent for in a hurry. Patient unconscious. Temp. 101° F.; pulse 120, irregular; breathing stertorous and of Cheyne-Stokes type. Inflammation of scalp worse, but no signs of pus; right eyelid so swollen that eyeball could not be examined; no retraction of head; no vomiting; rigidity of arms and legs, more marked on left side.

Incisions at once made in the œdematous scalp, and patient allowed to bleed from them freely. No pus was found. The head was then packed in ice, and 10 grs. of calomel given.

During whole of afternoon and night patient continued unconscious, with Cheyne-Stokes respiration (about 1 a.m. ceased breathing for intervals of fifteen to twenty seconds); chin had to be held forwards to allow patient to breathe; bowels open freely. Towards morning breathing improved, and patient could swallow.

At 9 a.m. on May 10th became conscious. In the

afternoon, when I was away, he got up and went out in the garden.

11th.—Much better. It can now be seen that parts of the scalp have been destroyed by the "Coachaline," and that sloughs are forming, varying in diameter from a quarter to three quarters of an inch.

During the next few days the sloughs began to separate, and it was seen that in places they extended the whole thickness of the scalp down to the pericranium. Patient's general condition satisfactory, but has attacks of violent paroxysmal pain in the head, especially at the back and in the right temple. When the œdema of right eyelid had sufficiently subsided, it was seen that the muscles supplied by the third nerve were paralysed; the pupil reacted to light.

The treatment throughout (except when patient was unconscious) was difficult, owing to the fact that he would keep neither dressing nor bandage on his head, and would insist on going out when he felt inclined. Mental condition somewhat affected.

27th.—Scalp healed. Ptosis of right eye and paralysis continue. General condition slowly improving.

June 18th.—Seen in consultation with Mr. Mules.

August 20th.—Can open and shut right eye. Wears tinted glasses; can see to drive, can read and write with difficulty with his old glasses. (*June 13th*, 1901.)

Mr. Devereux Marshall called Dr. Mules' attention to an interesting paper published by Dr. Head and Dr. Campbell in *Brain* last year, a notice of which appeared in the *Ophthalmic Review* in respect of herpes ophthalmicus. The authors had seen several cases, and had had the opportunity of performing post-mortem examinations on some of the patients at various intervals after the attack of herpes. In all the cases they found lesions in the Gasserian ganglion. They also pointed out the extremely epidemic nature of the cases: most of their cases came more or less together. They regarded it as an acute disease affecting some of the ganglia. In herpes ophthal-

micus the Gasserian ganglion was affected, and in herpes of the body other ganglia were involved. In these ganglia lesions of an inflammatory nature were found, and nearly always with hæmorrhage.

Dr. Aldren Turner referred to the infective nature of herpes zoster, and pointed out that motor nerves might be affected in a way similar to sensory nerves. In illustration of this, in addition to Mr. Mules's interesting experience, he mentioned the not-unknown sequence of Bell's palsy after herpes of the superficial cervical plexus, and of the paralysis and wasting of muscles which follow herpes of an ordinary mixed nerve, such as the sciatic. These facts seemed to argue in favour of the infective character of the pathological process underlying herpes zoster, which had been established by the recent researches of Drs. Head and Campbell.

Mr. Marcus Gunn asked whether there was any history of injury in the case. He said that some years ago he saw a patient at University College Hospital who had an injury to his head and symptoms of concussion. After the return of consciousness it was found that the right third nerve was paralysed, and that this eye was blind. Presumably, therefore, it was a case of fracture of the anterior part of the base, with injury to structures at the apex of the orbit. Some days later the patient developed herpes ophthalmicus, *i. e.* the fifth nerve had been involved in the injury, and from this lesion he had subsequently the mixed condition involving the motor and the sensory nerves. He also saw recently the case of a man who had a bullet injury to the anterior part of the skull, where the supra-orbital nerve was involved in the cicatrix. The patient had suffered from extreme neuralgia from this cause, and this was only finally relieved by dividing the supra-orbital nerve. Immediately following this operation the corresponding pupil became dilated, and remained so for some time. No cocaine was used. Possibly this effect was produced through the ciliary ganglion, but it was difficult to explain in any way. It seems to afford yet another

instance of the possible relation of the third and fifth nerves in injury.

The President said Mr. Gunn's case was a very curious one, and it was strange that the injury should have been followed by the herpes. Possibly the same thing might have happened in Mr. Mules's case.

Dr. Mules, in reply, said in the case of herpes there was no evidence of anything the matter with the eye, except the paralysis, and there was no history of injury.

8. *On Erythropsia.*

By W. H. R. Rivers, M.D.

Under the influence of bright illumination I have on several occasions observed a well-defined deep red border round white objects, especially when these are surrounded by deep black. The interest of this red border lies in the fact that it seems to furnish an intermediate link between the ordinary forms of erythropsia and a physiological phenomenon observed by Bidwell.

I have usually observed what may be termed "border erythropsia" when my eyes have been for some time illuminated by intense electric light, but I have on one occasion seen it in a well-marked form in strong sunlight. The border is a narrow, well-defined red band, which might, perhaps, on casual observation, be mistaken for chromatic aberration. I am, however, familiar with the latter, especially as it may be seen in artificial mydriasis, and I have no doubt that the red band is a different phenomenon.

The red border has sometimes lasted several minutes; in other cases it has been of short duration, sometimes lasting only a few seconds. In some cases it seems to

have been limited to that part of the field of vision corresponding to the part of the retina most strongly stimulated. In all cases I have observed it best in indirect vision, *i. e.*, with lateral parts of the retina.

I have failed to find any previous description of this phenomenon. Tait,* however, states that on waking from sleep, he has observed a lamp surrounded by a crimson ground for a second or two, and Rood† has observed a similar red halo after recovery from chloroform anæsthesia.

The form of erythropsia which I have described closely resembles the transient red border which has been described by Shelford Bidwell‡ as occurring round a brightly illuminated white area, when this area is suddenly exposed to an eye which has previously been for a time in the dark. This red border only lasts for about one-tenth of a second, but its colour-tone and general appearance are very much like the red border which I have described, and my attention was first attracted to the border in question on account of its resemblance to Bidwell's phenomenon.

The colour-tone of "border" erythropsia seems also to resemble that of the ordinary forms of erythropsia. I need hardly say that I have not observed the clinical form in my own person, but I have observed the closely-allied artificial erythropsia of Fuchs.§ This observer found that if one pupil be dilated when one is on snow-covered hills in bright sunshine, erythropsia occurs, which is apparently of the same nature as that which occurs clinically, especially after removal of the lens. The red colour is most easily seen on going from the bright sunshine into feeble illumination, when the white parts of a black and white surface will appear of a reddish colour, while the black parts become greenish.

* *Proc. Roy. Soc. Edinburgh*, vol. vii, p. 605, 1871–2.

† *Amer. Journ. Sci.*, ser. iii, vol. xiii, p. 32, 1877.

‡ *Proc. Roy. Soc.*, vol. lx, p. 368, 1896. See also *Curiosities of Light and Sight*, London, 1899, p. 202.

§ *Arch. f. Ophthalmol.*, Bd. xlii. Abth. iv, S. 207, 1896.

I have not repeated Fuchs's experiment at high altitudes, but at the low altitude of Cambridge I have obtained the same results as those described by Fuchs, by dilating the pupil of one eye with homatropine, and walking over snow-covered ground in bright sunlight. On going into dim illumination, the white squares of a black and white chess-board pattern were distinctly reddish, while the black squares were greenish. In one experiment, the red colour was limited to the edges of the white object, though in this case the red border was less well-defined than in the "border" erythropsia which I have described. So far as I can recollect, the general colour-tone of the red resembled that of "border" erythropsia, and of Bidwell's red border, although it was less intense.

It is probable that these different forms of red vision are closely related. The red border observed by me under Fuchs's experimental conditions seems to furnish an intermediate link between "border" erythropsia and the ordinary forms. The chief difference between clinical erythropsia and Fuchs's artificial erythropsia, on the one hand, and my "border" erythropsia and Bidwell's phenomenon, on the other hand, is that the red colour is diffuse in the one case, and well-defined in the other. The former phenomena occur, however, under conditions which lead to diffuse illumination of the retina, *viz.*, aphakia and mydriasis, and the difference between the definite border and the diffuse redness may well be explained by the different circumstances under which they arise.

The first object of this paper is to point out that Bidwell's red border, "border erythropsia," Fuchs's artificial erythropsia, and the erythropsia of aphakia, are probably closely related to one another, and that a satisfactory explanation of any one of these four phenomena should also be applicable to the other three.

I will next consider briefly the probable causation of erythropsia in general. Perhaps the most widely accepted view at present is that of Fuchs, namely, that the red colour is due to the entoptic vision of visual purple. There are great

difficulties in the way of this explanation. It is difficult to understand how we can see entoptically the colour of a substance which is contained within the sentient layer of the retina. Further, there is no doubt that in most of the recorded cases of clinical erythropsia, the red colour has been seen while the eye is still strongly illuminated, when the regeneration of visual purple assumed by Fuchs can hardly have begun.

It is far more likely that erythropsia is due to entoptic vision of another red substance within the eyeball, *viz.*, blood. There is one form of erythropsia,* which I have not so far mentioned, which is certainly due to the coloration of the light transmitted through the lateral walls of the eyeball. The transient red appearance of letters while reading in bright illumination is very familiar. Whenever I have observed this phenomenon, I have always found that I have been in strong lateral illumination, and double image observations, as in Fechner's side-window experiment, have shown a marked difference in the condition of colour-adaptation of the two eyes. In some cases I have observed all the print of a page distinctly red, and continuing in this colour for a long time. The red colour in this case is chiefly a contrast phenomenon, and may be seen on all dark objects on a lighter ground by an eye which has been exposed to strong lateral illumination. Bidwell mentions this appearance,† and regards it as identical with his red border phenomenon.

The idea that erythropsia is due to coloration of the light by blood in transmission through parts of the eyeball or lids has been advanced by Snellen‡ and by Koster.§

* Fuchs recognises that this form of erythropsia is due to lateral light. He also points out that red vision in some cases, as in hæmorrhage into the vitreous, or subhyaloid hæmorrhage, is due to blood. He regards the erythropsia accompanying aphakia and mydriasis, however, as something different.

† *Curiosities*, p. 206.

‡ *Arch. f. Ophthal.*, Bd. xliv, S. 19, 1897.

§ *Nederl. oogheelk. bijd.*, 1897.

It has, however, been shown by Birkhoff* that red vision may occur after gazing at a brightly illuminated surface for ten to fifteen minutes when all lateral light is excluded.

In addition to light passing through the eyelids, iris, and sclerotic, there are, however, two other ways in which light may become coloured red before it reaches the sensitive elements of the retina.

We know that under ordinary conditions of illumination light is reflected within the eyeball from the lateral parts of the retina on to its posterior part (the visibility of Purkinje's figures when seen by the ordinary method is due to such reflected light). The light reflected from the hyaloid membrane and from the internal limiting membrane will be colourless, but any light penetrating the retina and reflected from deeper layers will be coloured red by the blood in the anterior layers of the retina. It is noteworthy that the two chief conditions under which erythropsia occurs—*viz.*, mydriasis and aphakia—should be conditions in which the light so reflected would be very much increased in amount, owing to diffusion of light † within the eyeball. In Birkhoff's experiments, however, this source of erythropsia was also excluded, and Birkhoff ‡ has shown that erythropsia may occur when light is strictly limited to a small area of the retina.

The other source by means of which light reaching the retinal elements may be coloured red was not excluded by Birkhoff, and cannot be excluded by any experimental procedure. This source is the blood in the anterior layers of the retina—in the nerve-fibre layer, the ganglion-cell layer, the internal reticular layer, and the internal nuclear layer. These layers contain a thick meshwork of

* *Bijdrage tot de Kennis der Erythropsie*, Leiden, 1899.

† It is possible that the erythropsia which has been described in cases of opacities of lens and vitreous may be partly due to increased diffusion of light, though in most of the cases there has also been dilatation of the pupil (see Fuchs, loc. cit.).

‡ Loc. cit., p. 42, etc.

capillary blood-vessels, which furnishes a red layer through which all light reaching the retinal elements has to pass, except in the position of the fovea centralis.

There are three chief ways which in the light entering the eyeball may be coloured red: (1) by transmission through the eyelids, iris, and lateral walls of the eyeball; (2) by reflection from the internal lining of the eyeball; (3) by transmission through the anterior layers of the retina.

Under normal conditions the physiological mechanism is adapted to red.* We do not see objects red normally for the same reason that we do not see objects coloured when we wear coloured glasses long enough to allow the eye to become adapted to the colour.

Red vision may occur after the eye has been previously in comparative or absolute darkness and has ceased to be red-adapted, or it may occur when for any reason the coloration of the light entering the eye is more intense than under normal circumstances. The latter is the probable cause of the erythropsia which occurs after removal of the lens. There is little doubt that the lens absorbs light from the violet end of the spectrum, and in its absence rays from this end of the spectrum reach the retina in increased quantity, while the amount of ultra-violet light † passing into the vitreous is probably also increased. There is reason to believe that the light rays of short wave-length are those which especially produce sunburn of the skin, and it is highly probable that the excess of these rays reaching the retina sets up a hyperæmia of that membrane corresponding to that which they produce in the skin. If this should happen, the light reaching the rods and cones would be much more strongly coloured than in normal circumstances. Owing to the absence of the lens, a large amount of diffused light would be reflected from the lateral

* Schäfer's *Text-book of Physiology*, vol. ii, p. 1057.

† The importance of ultra-violet rays in the causation of erythropsia has been fully considered by Osswald in *Ueber Lichtsinnstörung und Erythropsie bei operierten Myopen, Beiträge zur Augenhlkde.*, Heft 45, S. 98, 1900; see also Fuchs, loc. cit.

walls, and would supplement the redness due to transmission through the anterior layers of the retina. All the circumstances under which the erythropsia of aphakia occurs are consistent with the idea that the red colour is due to entoptic vision of blood.

The same conditions are present in the artificial erythropsia of Fuchs. The dilated pupil allows more light to enter the eye, and this light is diffused throughout the vitreous more than in normal circumstances. It is well known that the light reflected from snow, especially at high altitudes, is most efficient in causing sunburn, and it is a small step to suppose that it may produce a similar condition of the retina.

The erythropsia which occurs after removal of the lens is often associated with hemeralopia.* The association of these conditions affords an additional argument against Fuchs's visual-purple theory, for in hemeralopia there is reason to believe † that there is interference with the formation of visual purple, and that this substance is formed in diminished quantity, and is therefore even less likely to be seen entoptically than under normal conditions. On the other hand, hemeralopia is probably associated with (and is a symptom of) a certain amount of retinal congestion or inflammation.

In the first part of this paper I have suggested that Bidwell's red border phenomenon may be closely related to erythropsia, and that any explanation of erythropsia in general should also be capable of application to this phenomenon. Bidwell supposes that his red border is due to special action of the nerve-fibres for red in the neighbourhood of an illuminated area. I have elsewhere ‡ pointed out that Kunkel's observation, that the sensory curve rises more rapidly for red than for other colours, is in favour of some such explanation.

* See Osswald, loc. cit., and Krienes, *Ueber Hemeralopie*, Wiesbaden, 1896.

† Schäfer's *Text-book of Physiology*, vol. ii, p. 1103.

‡ Ibid., vol. ii, p. 1075.

It seems, however, quite possible that the red border may be due to the coloration of the light entering the eye by blood. Bidwell's phenomenon occurs when the eye has previously been in darkness, *i.e.*, has lost to a certain extent its normal condition of red-adaptation. The red border may possibly be due to diffusion of red light round the illuminated area giving rise to a border, which is seen during the brief time which the retina takes in the process of again becoming adapted to red.

This explanation is perhaps highly hypothetical, but it is worth considering whether the phenomena of erythropsia may not give a clue to the explanation of what is otherwise a very obscure phenomenon.

There is one difficulty in the way of the view that erythropsia is due to entoptic vision of blood. The fovea centralis is free from blood-vessels, and it is only outside this region that the light transmitted through the anterior retinal layer will be tinged red. This is no difficulty for the clinical forms of erythropsia, for in these the light transmitted through the lateral parts of the eyeball and that reflected from the lateral walls of the retina will act on the fovea as well as on other parts of the retina. But in the artificial erythropsia seen by Birkhoff all sources of red coloration were excluded except that of the anterior layers of the retina, and it would seem that if the proposed explanation is correct, the erythropsia should in this case have been absent from the region of central vision. It is, however, only the centre of the fovea which is wholly free from capillaries, and a very small amount of diffusion of red light would obscure the corresponding part of the visual field. It is also possible that light reflected from the chorio-capillary layer behind the fovea might contribute and make the foveal region resemble the rest of the retina.

Owing to the absence of visual purple from the fovea, Fuchs's theory also requires the region of central vision to be colourless, and Fuchs * found that in some cases the

* Loc. cit., S. 230.

red colour did not occur, or was less marked in the region of central vision. According to his theory, this part of the visual field should be colourless under all circumstances, and in order to explain its presence Fuchs is obliged to make the fanciful assumption that the red colour is due to entoptic vision of visual purple in the processes of the cells of the pigment epithelial layer. If, however, erythropsia be due to entoptic vision of blood, it is only in those cases in which all sources of coloration but the anterior retinal layers are excluded that the area of the visual field corresponding to the fovea should be free from the red colour. The presence of erythropsia in the foveal region is a far greater difficulty for Fuchs's theory than it is for the view that erythropsia is due to entoptic vision of blood.

There is, however, a further difficulty in the way of the latter explanation. In many cases, especially in artificial erythropsia, the red colour is only seen on going into dim illumination after exposure to bright light. One is obliged to assume that the red colour is a positive after-image effect of the red stimulation to which the eye has been exposed, and that this positive after-image effect is seen in dim illumination, while the effect of the actual red stimulation remains below the threshold, owing to the simultaneous strong excitation of the non-chromatic processes. Such an assumption seems somewhat forced, but is consistent with much that we know of visual sensations. Colour effects may often be noticed in an after-image which have passed unnoticed in the effect of the original stimulation, and erythropsia occurs under just those conditions which favour the occurrence of positive after-image effects. In some cases of artificial erythropsia, on the other hand, the red coloration may be seen in bright light, and in clinical erythropsia this is apparently the rule, especially in the more marked cases.

It is in agreement with the idea that erythropsia is, in some cases, an after-image effect, that the red vision may

often be associated with green vision. Erythropsia may be preceded by, or followed by, green vision; while in other cases some part of the field of vision may be red, while others are green. The positive after-image effect may in some cases be associated with a negative after-image effect, while in other cases the red and green may be seen as contrast colours.

It is interesting that Bidwell's red border may also, under certain circumstances, be transformed into a green border, and Bidwell * has shown that this green border—which is seen with very bright illumination—is an after-image of the red, the after-image being seen while the colour of which it is an after-image is not seen. This fact provides another point of resemblance between Bidwell's red border and the ordinary forms of erythropsia.

(*June 13th*, 1901.)

Mr. Swanzy thought there was clinically still another form of erythropsia not included in Dr. Rivers's paper. He had himself, on more than one occasion, distinctly experienced it, and it seemed not quite to agree with the theory put forward by Dr. Rivers, for it especially affects the portion of the field of vision corresponding with the macula lutea. The first time he noticed it it gave him great alarm, for it occurred while examining a patient about whose case he was anxious, and whom he had to go into the country to see. The light supplied to him for the examination was a very good duplex paraffin lamp. The flame, however, was not burning quite satisfactorily, and he adjusted the wick by means of the screw, watching the flame the while. On then looking at the patient's eye he was astonished to find what appeared to be a large hæmorrhage in the anterior chamber. He shut his eyes for a moment or two, and on looking again after a little time there was no hæmorrhage. He saw that gentleman on a subsequent occasion, when the same thing occurred. A similar event, but happening in a different

* *Proc. Roy. Soc.*, vol. lxi, p. 269.

way, took place when he had begun to read a book immediately after looking at a bright incandescent gas mantle. The black letters of the book were red where he fixed them, but all the letters in the periphery of the field were black.

Dr. Edridge-Green thought that the remarks of Mr. Swanzy showed that Dr. Rivers had not proved his point. He (Dr. Green) had devoted considerable attention to the subject, and had found that the red object was seen clearly with the fovea. The fact that seemed to be strongly against the view that it might be due to the blood was, that it was much more difficult, as Bidwell mentioned, to obtain a rose-coloured positive after-image with red than with any other light. It was most easily seen after green light.

Dr. Rivers, in reply, said the phenomenon referred to by Dr. Edridge-Green was the recurrent image, also described by Bidwell. The red border mentioned in the paper had no connection with this. With regard to the form of erythropsia in which black letters appeared red, he omitted mention of that because of the lateness of the hour. He had often observed it, and had always found that it was due to the influence of lateral light passing through the sclerotic. The other phenomenon observed by Mr. Swanzy, that of apparent hæmorrhages, was an after-image effect, which was a different phenomenon altogether, and should not be confused with erythropsia.

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9. *A case of blindness resulting from the administration of salicylate of sodium.*

By Simeon Snell.

On April 2nd, 1901, I was asked by a medical friend to see a young girl, a patient of his, who had suddenly lost the sight in both eyes. On visiting the house I found the patient was æt. 9 years, and learnt the following history.

On March 27th she had attended school as usual. The next day (28th), as she complained of pains in her joints, she was kept at home. On the 29th my friend was sent for, and found her suffering from acute articular rheumatism, affecting all the joints to some degree, but especially those on the left side. Salicylate of sodium was ordered in five-grain doses, to be administered every three hours. There is nothing to report as to the next two days. During the night March 31st to April 1st she was much disturbed and restless. Her mother was sitting up with her, and frequently spoke to her. At about 7.30 a.m. she awoke from a sleep and spoke of everything being very dark in front of her. Before this she had made no reference to her sight. Later on in the morning, she mentioned that it was so dark that she would like the gas to be lighted. At my friend's visit, between eleven and twelve o'clock, he found her unable to distinguish light from dark. The next day, April 2nd, when I saw her, she was suffering considerably from the articular rheumatism. She was perfectly blind, there being, as far as I could make out, an entire absence of light-perception. The sensation of the cornea and the surface of the eyeball were unimpaired. Both pupils were rather dilated, but responded somewhat to light. The media were clear, and an ophthalmoscopic examination, made under difficulties, was sufficient to indicate that the fundus was normal in both eyes. There was no deafness, nor was tinnitus complained of. The next day I visited the patient again, and her general condition was very much worse. She was suffering from pericarditis with endocarditis, and it was evident that she would not survive long. Nothing abnormal could be detected in either fundus. There was no return whatever of vision. She died the same night at 11.30. A post-mortem was asked for, but refused.

Remarks.—It has been mentioned that the intended dose of salicylate of sodium was five grains, but the quantity actually administered was ten grains at each dose. The drug was discontinued on the morning of April 1st, at the

time she first complained of her sight having failed. In making a calculation it would appear that in about sixty hours she must have taken about 140 to 160 grains of the salicylate of sodium.

I do not think there can be any question that the salicylate of sodium was the cause of blindness in this case. A limited number of similar cases have been recorded. De Schweinitz, in his classical work on *Toxic Amblyopias*, refers to the instances on record. He mentions particularly a case by Gatti. The patient was a robust girl of sixteen, to whom the salicylate of sodium was administered for articular rheumatism. Eight grammes divided into ten-grain doses, a dose being taken hourly, was administered. After the last dose she fell asleep, and woke entirely blind. There was well-marked mydriasis; the optic papillæ were normal. Ten hours later the patient could count fingers, and twenty-four hours after the blindness began, vision was restored.

My case bears a striking resemblance to the above. Each patient was a young girl—mine being the younger,—and each was the subject of articular rheumatism; but whereas recovery of sight took place in Gatti's, my case terminated fatally. (*July 5th*, 1901.)

10. *A note with regard to the surface of the normal cornea.*

By H. Herbert, Major, I.M.S., Bombay.

I wish to draw attention to a fact of no great importance, but yet one worthy of notice and comment. If the normal cornea be partially dry by exposure, the surface, which appears at first absolutely and uniformly smooth, soon shows a greater or less degree of epithelial defect, which is ordinarily completely filled up and obscured by lacrymal fluid. In exposing the cornea I have employed the finger of an assistant to elevate the upper lid sufficiently to

remove it from contact with the eyeball, while the patient looked steadily to the front; or a speculum was introduced, generally after cocaine instillation. Focal illumination and the corneal *loupe* are necessary to see the defects clearly, and the observer needs to move his head, in order to catch the bright reflex from different parts of the corneal surface. I have examined without cocaine over thirty eyes of patients with perfectly normal conjunctiva and cornea, or with only very slight conjunctival injection. Usually only a portion of each cornea was inspected, owing to the lacrymation induced by the exposure and the discomfort to the patient. Depressions generally began to appear within half or one minute, in some cases within a few seconds; but in one case not till after three minutes and some seconds, apparently owing to some defect in the arrangements for drying. In no case were depressions entirely wanting, though they varied much in number and extent. In some cases only one or two small rounded pits were seen; in others very numerous closely set pits; in others, besides pitting, one or more grooves, generally radial, were found; and in a few instances one or more extensive and irregular depressed patches were seen. The depressions all appeared sharply-defined. They showed themselves gradually increasing in number and extent for perhaps two or three minutes, as the drying of the surface went on. The portion of cornea perhaps oftenest affected by pitting was the part commonly exposed in the palpebral aperture; the comparatively rare large patches generally lay close to the corneal margin. The floors of the depressions are always stippled, and can often be seen in the non-illuminated portions of the cornea as grey points and patches against the background of iris; the stippling is, of course, due to the irregular surface presented by the deeper cells exposed. To make sure that the appearances represented real defects of surface epithelium, and were not simply the expression of irregular drying, I several times had the drying surface wiped with a pencil of lint by an assistant while under observation.

I found that second and third exposures often, but not always, revealed considerably more epithelial defect than the first attempt; and this was especially so if cocaine had been used in the intervals. After cocaine instillation alone, or after previous exposure and cocaine, there was often superficial exfoliation of epithelium in strips and patches, numerous, extensive and irregular, producing areas perhaps scarcely at all stippled and only very slightly depressed. But that the deeper pittings, grooves, and patches seen on first exposures had existed previously, and were not manufactured by the drying, was shown by their almost immediate appearance in many cases, and by their remaining the same after repeated short exposures.

It may be that these epithelial irregularities are not to be found so constantly in Europe. All of my observations have been conducted in Bombay on natives of the country, except in one case a European; they were made in the rainy season. Most of the subjects were adults, owing to the difficulty of keeping children's eyes still; but one examinee was only ten years of age, and a few others but slightly older. A number of variously inflamed or otherwise abnormal eyes were also similarly examined, without cocaine when possible. It was found that on the whole the slighter forms of conjunctivitis and (localised) keratitis were accompanied by an increase in the amount of pitting. In one acute catarrhal conjunctivitis—ocular membrane a good deal affected—the superficial epithelium appeared to have exfoliated from the whole corneal surface. I have seen lately a number of cases of superficial punctate keratitis (unilateral). The points all or nearly all appear a little elevated at first; but on exposure some of the points begin to dry early, and then appear as stippled depressions. Both the elevation and the depression are largely the result of irregularity in the layer of fluid on the surface. After cocaine instillation and fluorescein staining, a careful drying of the surface, with lint if necessary, seems to show in some cases both the physiological sharply-cut unstained pits distinguishable from others pathological, stained, and less sharply defined.

(Some of the stained points—the smaller ones I think—appear still slightly raised.)

Apart from the distinction from pathological processes here referred to, the only point of interest in connection with these physiological depressions is the evenness with which they are levelled up by lacrymal fluid, so as not to interfere with the refraction of the eye. (*March 14th*, 1901.)

XVI. APPENDIX.

The following communications were also brought before the Society during the session.

1. Double cataract rapidly developing after typhoid fever contracted in South Africa.
   E. Kenneth Campbell (October 18th, 1900).

2. Large dermoid growth of cornea in an ox's eye.
   W. T. Lister (December 13th, 1900).

3. Nævus of orbit.
   Holmes Spicer (December 13th, 1900).

4. Tubercle beneath ocular conjunctiva.
   R. Marcus Gunn (December 13th, 1900).

5. Specimen of primary sarcoma of orbit.
   A. W. Stirling (January 31st, 1901).

6. De Yeng's corneal microscope.
   Kenneth Scott (March 14th, 1901).

7. A new ophthalmoscope.
   R. W. Doyne (March 4th, 1901).

8. Periscopic glasses.
   A. S. Percival (May 2nd, 1901).

9. A thorn removed after two months' sojourn in the eyeball.
   J. B. Story (May 2nd, 1901).

10. Chancre of the lids.
    C. Wray (May 2nd, 1901).

11. Case of detached retina cured by operation.
    C. Wray (May 2nd, 1901).

12. ? Exostosis of frontal sinus.
C. Wray (May 2nd, 1901).

13. Case of sympathetic ophthalmitis (?) occurring nearly ten years after enucleation of the injured eye.
J. Hern (June 13th, 1901).

14. Growths of conjunctiva in an old man; for diagnosis.
A. Bronner (June 13th, 1901).

15. Tubercle of the iris.
Inglis Taylor (June 13th, 1901).

16. "Hole" in the retina.
R. W. Doyne (June 13th, 1901).

17. Gumma of the orbit.
C. Wray (July 5th, 1901).

18. A peculiar form of vitreous opacity.
Inglis Taylor (July 5th, 1901).

REPORT OF THE COUNCIL.

Read at the Annual General Meeting of the Society, July 5th, 1901.

The Council have the pleasure to report that the past Session has been highly successful, both as regards the number and the quality of the communications and the character of the discussions; as well as in the number of new members who have joined the Society. Seventeen new members have been added to the roll; four have resigned; and two, Messrs. Davidson of Swansea, and Bowater Vernon of London, have died.

The Council have further pleasure in reporting that Prof. Fuchs, of Vienna, has accepted their invitation to deliver the Bowman Lecture in 1902.

As the result of a recent census of resident members regarding their views upon a suggested alteration in the days of ordinary meetings from Thursday to Friday, the Council have decided to hold the ordinary meetings in November and March of next session upon Friday instead of Thursday evening.

The Council have also decided to more strictly enforce the regulations governing the clinical evenings, especially as regards the number of patients exhibited; and to postpone their discussion until 9 p.m.

In reply to an invitation from the Congress on Tuber-

culosis, to be held this month in London, that the Society should send a delegate, the Council requested the President to be their representative, and he has kindly consented to act in this capacity.

The thanks of the Society are due to Mr. Nettleship for his valuable gift of over three hundred pamphlets to the library. These were selected from a much larger number as being especially suitable for the Society's library, and they contained no duplicates. Mr. Nettleship has also presented several instruments of interest. Books have also been received from Prof. Hirschberg, Dr. Kerr, and Mr. McGillivray.

The Treasurer's report and balance-sheet are in the hands of members, and show a satisfactory financial condition.

ACCOUNT OF RECEIPTS AND PAYMENTS
OF THE
OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

Receipts.

1900–1901.	£	s.	d.
To Balance at Bankers, June 27th, 1900	168	8	1
,, Subscriptions	292	19	0
,, Entrance Fees (12)	25	4	0
,, Composition Fees (7)	54	9	0
,, Trustees of the Bowman Fund	50	0	0
,, Messrs. Churchill, Sale of 'Transactions'	28	4	6
,, Dividends:			
East India Railway B Annuities £17 11 8			
Ramsgate Corporation 3 per cent. Stock 11 5 7			
	28	17	3
	£648	1	10

Investments:
£20 East India Railway B Annuities.
£420 Ramsgate Corporation 3 per cent. Stock.

(Signed) JOHN ABERCROMBIE, *Treasurer.*

June 26th, 1901.

Payments.

1900–1901.	£	s.	d.
By Rent	80	0	0
,, Insurance	0	12	6
,, Printing Vol. XX of 'Transactions'	180	13	0
,, Illustrations for do. £99 4 6			
Less contributions from authors 29 15 0			
	69	9	6
,, Reporting Meetings	8	2	3
,, Hire of microscopes	3	3	0
,, Sub-Librarian	10	10	0
,, Refreshments at Meetings	8	7	0
,, Petty Cash Disbursements	3	16	3
,, Bankers' Charges	0	5	7
,, Purchase of Ramsgate Corporation Stock	67	3	4
,, Balance, June 26th, 1901	215	19	5
	£648	1	10

Examined, compared with vouchers, and found correct.

(Signed) { L. VERNON CARGILL, / C. DEVEREUX MARSHALL, } *Auditors.*

INDEX.

PAGE

A capsulotomy knife 143
A case of blindness resulting from the administration of salicylate of sodium 306
—— of bullet injury of the occipital lobes, with loss of the lower half of each visual field 123
—— of congenitally imperfect separation of iris from back of cornea 166
—— of high hypermetropia 171
—— of intra-ocular hydatid 89
—— of pulsating exophthalmos following injury to the head . 135
A diagrammatic model intended to assist in the teaching of ocular refraction 142
A large area of retinal pigmentation below the disc . . . 169
A note with regard to the surface of the normal cornea . 308
A portable refractometer 156
A rare example of persistent capsulo-pupillary membrane . 168
A trichromic or three-unit case of colour-blindness . . 181
Ætiology of lamellar cataract 43
An apparatus for the performance of vibration massage, as adapted for the treatment of eye diseases . . 158
An encapsulated sarcoma of the right orbit . . 204
An unusual form of choroiditis 68
Anderson (Tempest), discussion on superficial choroidal atrophy 68
Appendix 312

Batten (Rayner D.), discussion on an unusual form of choroiditis 69
—— injury to an eye by direct sunlight 70

PAGE

Batten (Rayner D.), serous iritis with nodular formations in the anterior chamber 35
—— —— with persistent tachycardia . . . 36
Bennett (Norman G.), ætiology of lamellar cataract . 43
Berry (G. A.), discussion on case of probable frontal mucocele 97
—— —— on "eclipse-blinding". . . . 79
—— on so-called paresis of divergence . . . 268
—— on two rare forms of non-paretic, non-concomitant squint 284
Blair (Charles), a portable refractometer . . . 68
—— an unusual form of choroiditis . . . 156
Bokenham (T. J.), discussion on "eclipse blinding" . 78
Bronner (A.), discussion on case of probable frontal mucocele 97
Buchanan (Leslie), case of carcinoma of corneo-scleral margin 205
—— cyclitis: a study of the inflammatory exudations into the vitreous body in cases of cyclitis . . . 208
Bullar (J. F.), notes of a case of membranous conjunctivitis . 9
Bullet wound of occipital region 127

Carcinoma of corneo-scleral margin . . . 205
Cartwright (E. H.), discussion on peritomy . . 153
Case of bullet wound of orbit followed by blindness of both eyes 121
—— of probable frontal mucocele . . . 95
Cases of blindness from gunshot injury of the orbit . . 102
Cataract, lamellar, ætiology of 43
Central choroiditis, simulating a neoplasm . . 80
Choroid and retina, diseases of 66
Choroidal disease of unusual appearance (? tuberculous) . 87
Choroiditis, an unusual form of 68
—— central, simulating a neoplasm . . . 80
—— shown in an early stage in December, 1898 . . 81
Collins (E. Treacher) and Ford (A. Vernon), bullet wound of orbit 121
—— and Hinnell (J. S.), diminished tension and the appearances of optic neuritis persisting for nine months after a wound of the orbit by a shot from an air-gun . . 100
—— discussion on blindness from gunshot injury of the orbit 118
—— —— on "eclipse-blinding". . . . 77
—— —— on pulsating exophthalmos . . . 139
—— —— on the ætiology of lamellar cataract . . 64

PAGE
Collins (E. Treacher), report on epithelioma of the cornea . 25
—— —— on sarcoma of orbit 205
Coloboma of eyelid with deformity of auricle and dermoid growth 173
—— of optic disc 171
Colour-blindness 181
Colour-sense, evolution of the 182
Concussion injuries of the left eyeball . . . 122
Congenital conditions 165
—— fibro-vascular tumours of eyeball . . . 178
—— malformation of eyelids with symmetrical lacrymal fistulæ 165
Conjunctiva, dermoid of. 172
—— diseases of 1
—— melanotic sarcoma of 13
—— primary chancre of 3
—— tuberculosis of 1, 7
Conjunctival bridges and pouches . . . 16
—— growth (? pinguecula) 5
Conjunctivitis, membranous 9
Cornea, a note with regard to the surface of the normal . 308
—— diseases of the 24
—— epithelioma of 24
—— growth on 27
—— rare form of symmetrical opacities . . . 32
—— spreading opacity of, following herpes frontalis . 31
Critchett (Anderson), a case of bullet injury of the occipital lobes with loss of the lower half of each visual field . 123
Cropper (J.), discussion on peritomy . . . 154
Cross (F. R.), congenital fibro-vascular tumour of eyeball 178
Crystalline lens, diseases of 43
Cyclitis 208

Diminished tension persisting for nine months after a wound of the orbit 100
Dodd (H. Work), a case of high hypermetropia . . 171
—— nævoid condition of retina 82
—— voluntary bilateral nystagmus . . . 244
Double coloboma of right lens 179
—— frontal sinus mischief simulating symmetrical exostoses in the neighbourhood of the lacrymal sac . . 94

PAGE

Double coloboma, temporal hemianopia with slight optic neuritis and hemianopic pupil reaction. ? Hæmorrhage into optic nerve-sheath at chiasma. ? Gumma . . 286
Drake-Brockman (Herbert E.), new pattern of eye speculum 162

"Eclipse-blinding" 70
Edridge-Green (F. W.), a trichromic or three-unit case of colour-blindness 181
—— discussion on erythropsia 306
—— —— on primary chancre of retro-tarsal fold of upper lid 4
—— —— on superficial choroidal atrophy . . 67
—— the evolution of the colour-sense . . . 182
Enucleation and growth of the orbit . . . 258
—— sympathetic disease following . . . 37
Epithelioma of the cornea 24
Erythropsia 296
Exophthalmos, pulsating 135
Eye speculum, new pattern 162

Fisher (J. Herbert), a case of congenitally imperfect separation of iris from back of cornea; rise of tension induced by use of a mydriatic 166
—— bullet wound of occipital region . . . 127
—— epibulbar sarcoma 29
—— subretinal cystic swelling at centre of fundus . . 83
—— tuberculosis of the conjunctiva . . . 1
Flemming (Percy), sections showing opaque nerve-fibres in the retina 200
Ford (A. Vernon) and Collins (E. Treacher), case of bullet wound of orbit followed by blindness of both eyes . 121
Frost (W. Adams), coloboma of eyelid with deformity of auricle and dermoid growth 173
—— discussion on retinal pigmentation . . . 170

Goldsmith (H.), discussion on growth on cornea . . 28
Griffith (John), discussion on ætiology of lamellar cataract 64
—— double frontal sinus mischief . . . 94
—— microscopical specimen of an osteosarcoma of the orbit 201
Grimsdale (Harold B.), congenital malformation of eyelids with symmetrical lacrymal fistulæ . . . 165
—— discussion on ætiology of lamellar cataract . . 64

PAGE

GRUBER (R.), discussion on blindness from gunshot injury of the orbit 117
—— —— on influence of eyeball on growth of orbit . 267
GUNN (Donald), discussion on blindness from gunshot injury of the orbit 118
GUNN (R. Marcus), discussion on paralysis of third nerve . 295
—— recent central choroidal disease of unusual appearance . 87
Gunshot injury of the orbit 102

HARMAN (N. B.), discussion of conjunctival bridges and pouches 23
—— "Hole" at the macula 88
HARTRIDGE (Gustavus), a capsulotomy knife . . 143
—— case of central choroiditis, simulating a neoplasm, watched for fourteen years 80
—— case of choroiditis shown in an early stage in December, 1898 81
—— discussion on blindness from gunshot injury of the orbit 117
—— —— on corneal opacity following herpes frontalis . 31
—— —— on the orthoptic treatment of convergent squint . 257
—— nævoid condition of the vessels of the disc . . 83
HAWTHORNE (C. O.), discussion on an unusual form of choroiditis 70
HERBERT (H.), a note with regard to the surface of the normal cornea 308
—— conjunctival bridges and pouches; union of retro-tarsal folds 16
—— hypertrophy and ossification of tarsus . . 202
HERN (J.), discussion on peritomy . . . 153
Herpes frontalis followed by spreading corneal opacity . 31
Hess's operation, modified 174
HINNELL (J. S.) and COLLINS (E. Treacher), diminished tension and optic neuritis persisting for nine months after a wound of the orbit by a shot from an air-gun . . 100
Histology 200
"Hole" at the macula 88
HUDSON (Ainslie), an apparatus for the performance of vibration massage 158
Hydatid, intra-ocular 89
Hypermetropia, high 171
Hypertrophy and ossification of tarsus . . . 202

PAGE
Injuries of the eye and orbit 98
Injury to an eye by direct sunlight; "eclipse-blinding" . 70
Intra-ocular hydatid 89
Iris and ciliary body, diseases of 35

James (G. Brooksbank), a large area of retinal pigmentation below the disc 169
—— a rare example of persistent capsulo-pupillary membrane 168
Jessop (Walter H.), case of probable frontal mucocele . 95
—— discussion on an unusual form of choroiditis . . 69
—— —— on "eclipse-blinding" . . . 78
—— sequel to case of tuberculosis of conjunctiva . . 7

Knife, a capsulotomy 143

Lamellar cataract, ætiology of 43
Lang (William), dermoid of conjunctiva with fissure of lid . 172
—— new growth on conjunctiva and cornea . . 8
—— rupture of the lamina cribrosa and optic nerve-fibres at the papilla 98
Lawford (J. B.), concussion injuries of the left eyeball . 122
—— discussion on "eclipse-blinding" . . . 76
—— further notes of a case of melanotic sarcoma of the conjunctiva 13
Lawson (Arnold), Thiersch grafting for xerophthalmia . 157
Lens, coloboma of 179
Lid, fissure of 172
Lightning, two cases of eye injury due to . . . 139
Lister (W. T.), spreading opacity of the cornea following herpes frontalis 31
Little (David), discussion on case of probable frontal mucocele 97
—— —— on intra-ocular hydatid . . . 92

Mackay (George), note on a streptothrix mass (? actinomyces) removed from the right inferior canaliculus . 207
Macula, "hole" at the 88
Marshall (Devereux), discussion on membranous conjunctivitis 12
—— —— on paralysis of third nerve . . . 294
—— —— on secondary carcinoma of both eyes . . 84
—— —— on sympathetic disease following enucleation . 40

PAGE
Microscopical specimen of an osteosarcoma of the orbit . 201
Miscellaneous 244
Morton (A. Stanford), double coloboma of right lens . 179
—— growth on cornea 27
Mules (P. H.), double symmetrical opacities of cornea (rare form) removed by operation 32
—— paralysis of third nerve with unusual complications . 292

Nævoid condition of retina 82
—— of the vessels of the disc with nævus of the eyelid and face 83
Nettleship (Edward), cases of blindness from gunshot injury of the orbit 102
—— discussion on intra-ocular hydatid . . . 92
—— —— on pulsating exophthalmos . . . 139
—— —— on superficial choroidal atrophy . . 67
—— postscript to case of intra-ocular hydatid . . 90
New growth on conjunctiva and cornea . . . 8
Note on a streptothrix mass (? actinomyces) removed from the right inferior canaliculus . . . 207
Notes of a case of membranous conjunctivitis . . 9
Nystagmus, voluntary 244

On erythropsia 296
On peritomy in episcleritis, iritis, herpes ophthalmicus, and purulent ophthalmia 146
On so-called paresis of divergence . . . 268
On two rare forms of non-paretic, non-concomitant squint . 284
Operations, instruments, and appliances . . . 142
Optic disc, coloboma of 171
Orbit and sinuses, diseases of 94
—— encapsulated sarcoma of 204
—— osteosarcoma of 201
Ormond (Arthur W.), primary chancre of retro-tarsal fold of upper lid 3
Orthoptic treatment of convergent squint . . . 245

Paralysis of third nerve with unusual complications . 292
Parasites 89
Parsons (J. Herbert), coloboma of optic disc . . 171
—— discussion on the evolution of the colour-sense . 199
Pathological anatomy 201
Peritomy 146

PAGE
Persistent capsulo-pupillary membrane . . . 168
President, discussion on a capsulotomy knife . . 143
—— —— on a streptothrix mass removed from lower canaliculus 208
—— —— on blindness from gunshot injury of the orbit . 117
—— —— on case of choroiditis, shown in an early stage in December, 1898 81
—— —— on cyclitis 243
—— —— on growth on cornea 28
—— —— on influence of eyeball on growth of orbit . 267
—— —— on lamellar cataract 64
—— —— on membranous conjunctivitis . . . 11
—— —— on new pattern of eye speculum . . 164
—— —— on paralysis of third nerve . . . 296
—— —— on peritomy 152
—— —— on primary chancre of retro-tarsal fold of upper lid 3
—— —— on pulsating exophthalmos . . . 139
—— —— on spreading opacity of the cornea following herpes frontalis 31
—— —— on superficial choroidal atrophy . . 67, 68
—— —— on symmetrical corneal opacities (rare form) removed by operation 32
—— —— on sympathetic disease following enucleation . 40
—— —— on the evolution of the colour-sense . . 199
—— —— on tuberculosis of the conjunctiva . . 2
Primary chancre of retro-tarsal fold of upper lid . . 3

Recent central choroidal disease of unusual appearance (? tuberculous) 87
Recording colour perimeter 144
Refractometer, a portable 156
Report of committee on conjunctival growth (? pinguecula) . 6
Retina and choroid, diseases of 66
—— nævoid condition of 82
Rivers (W. H. R.), on erythropsia . . . 296
Rockliffe (W. C.), conjunctival growth (? pinguecula) . 5
—— secondary carcinoma of both eyes following scirrhus of breast 84
Roll (G. W.), two cases of congenital ptosis upon which a modified Hess's operation had been performed . . 174
Rupture of the lamina cribrosa and optic nerve-fibres at the papilla 98

PAGE
Sarcoma, epibulbar 29
Secondary carcinoma of both eyes following scirrhus of breast 84
Sections showing opaque nerve-fibres in the retina . . 200
Sequel to case of tuberculosis of conjunctiva . . 7
Serous iritis with nodular formations in the anterior chamber 35
with persistent tachycardia 36
Snell (Simeon), a case of blindness from the administration of salicylate of sodium 306
—— an encapsulated sarcoma of the right orbit . . 204
—— discussion on primary chancre of retro-tarsal fold of upper lid 4
—— —— on sympathetic disease following enucleation . 40
—— epithelioma of cornea 24
Sodium salicylate, blindness from . . . 306
Speculum, eye, new 162
Spicer (W. T. Holmes), spring catarrh in the quiescent stage 12
Spreading opacity of the cornea following herpes frontalis . 31
Spring catarrh in the quiescent stage . . . 12
Squint, the treatment of, in young children . . 245
Stephenson (Sydney), discussion on membranous conjunctivitis 11
—— wound of eyeball with prolapse of iris; enucleation of eyeball twenty days after the accident, followed fifty-four days later by sympathetic disease of the other eye; recovery 37
Strabismus fixus 284
Subretinal cystic swelling at centre of fundus . . 83
Superficial choroidal atrophy, without subjective symptoms, in a member of a family subject to night-blindness . 66
Swanzy (H. R.), discussion on erythropsia . . 305
Sympathetic disease 37

Tachycardia with serous iritis 36
Tarsus, hypertrophy and ossification of . . . 202
Taylor (Johnson), discussion on "eclipse-blinding" . 79
—— —— on peritomy 153
—— —— on tuberculosis of the conjunctiva . . 2
Teale (T. Pridgin), on peritomy in episcleritis, etc. . . 146
The determination of the influence of the eyeball on the growth of the orbit by experimental enucleation of one eye in young animals 258
The orthoptic treatment of convergent squint in young children 245

PAGE
Thiersch grafting 30
—— for xerophthalmia 157
Thompson (A. Hugh), double temporal hemianopia with slight optic neuritis and hemianopic pupil reaction. ? Hæmorrhage into optic nerve-sheath at chiasma. ? Gumma 286
—— superficial choroidal atrophy, without subjective symptoms, in a member of a family subject to night-blindness 66
Thompson (Tatham), discussion on blindness from gunshot injury of the orbit 120
—— —— on case of probable frontal mucocele . . 96
Thomson (W. Ernest), the determination of the influence of the eyeball on the growth of the orbit by experimental enucleation of one eye in young animals . . 258
Tomlinson (J. H.), recording colour perimeter . . 144
Tubercle of choroid 87
—— of conjunctiva 1, 7
Turner (W. Aldren), discussion on paralysis of third nerve 295
Two cases of congenital ptosis upon which a modified Hess's operation had been performed . . . 174
—— of eye injury due to lightning . . . 139

Vibration massage 158
Vitreous body, exudations into, in cases of cyclitis . . 208
Voluntary nystagmus 244

Walker (H. Secker), a model for the teaching of ocular refraction 142
—— discussion on peritomy 154
Wells (A. P.), discussion on case of probable frontal mucocele 96
Wood (David J.), a case of intra-ocular hydatid . . 89
—— —— pulsating exophthalmos following injury to the head 135
Worth (C.), the orthoptic treatment of convergent squint in young children 245

Xerophthalmia, Thiersch grafting for . . . 157

Yarr (M. T.), two cases of eye injury due to lightning . 139

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